A Study of

ACUTE INTESTINAL OBSTRUCTION

Dissertation submitted to

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CERTIFICATE

This is to certify that this dissertation in **"ACUTE INTESTINAL OBSTRUCTION"** is a work done` by **DR. TAMIL MUTHU.M,** under my guidance during the period 2010-2012. This has been submitted in partial fulfillment of the requirements for the award of M.S. Degree in General Surgery (Branch – I) by the Tamil Nadu Dr. M.G.R. Medical University, Chennai – 32.

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DECLARATION

I hereby solemnly declare that the dissertation titled “A Study of Acute Intestinal Obstruction was done by me, under the guidance of PROF. DR. M. SEKARAN, M.S., at Government Rajaji Medical College and Hospital, Madurai, during the period 2010-2012. The dissertation is submitted to the Tamil Nadu Dr. M.G.R. Medical University towards the partial fulfillment of the requirements for the award of M.S. DEGREE IN GENERAL SURGERY (BRANCH – I).

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CONTENTS

INTRODUCTION

Acute intestinal obstruction is one of the most common surgical emergencies. A constellation of conditions causing bowel obstruction at different levels and by different mechanisms can present with similar clinical features. Its early recognition and aggressive treatment can prevent irreversible ischemia and transmural necrosis, thereby decreasing mortality and long-term morbidity. Despite many recent advances in our diagnostic and treatment modalities, the morbidity and mortality due to acute intestinal obstruction remains high. Earlier, obstructed hernia was considered to be the most common cause of acute intestinal obstruction. Now adhesions are the most common cause world wide, but in developing countries hernia still accounts for most cases.

In this study, we will discuss about the age and sex incidence, various causes, clinical features, management and the outcomes of acute intestinal obstruction in our hospital.

AIM OF STUDY

1. To study the age and sex distribution of acute intestinal obstruction.

2. To study the various etiologies of acute intestinal obstruction.

3. To discuss the various clinical manifestations of acute intestinal obstruction.

4. To discuss the various treatment modalities for acute intestinal obstruction.

5. To study the outcomes of patients with acute intestinal obstruction.

REVIEW OF LITERATURE

DEFINITION:

Intestinal obstruction is defined as partial or complete blockage of either small or large intestine or both resulting in failure of intestinal contents to pass beyond the point of obstruction. In other words intestinal obstruction occurs when the normal propulsion and passage of intestinal contents does not occur. It can be either mechanical or functional obstruction, which is also called as Ileus.

HISTORICAL BACKGROUND

History of intestinal obstruction dates back to third or fourth centuries. Hippocrates was the one who recognized, described and treated intestinal obstruction. During this period Praxagoras was the first surgeon made an enterocutaneous fistula to relieve intestinal obstruction. However until late 1800's non-operative managements like hernia reduction, ingestion of laxatives and heavy metals was practiced in most parts of the world. [1]

In seventeenth century, Heister did a resection of strangulated bowel with diversion. He was followed by Pillare who did caecostomy for rectal malignancy. In 19th century Henri Hartmann described Hartmann's procedure.

Recent advances made in the field of minimally invasive surgery have shown that laparoscopic approach for acute intestinal obstruction was found to be effective in selected group of patients.

SURGICAL ANATOMY

SMALL INTESTINE:

The small intestine is considered to be the longest part of the gastrointestinal tract. It extends from the Duodenum to the ileocecal junction. Small intestine is approximately 6-7 metres long. It consists of the duodenum, the jejunum, and the ileum.

1. DUODENUM:

It is the first part of the small intestine. It is 20-25 cm long. It is the widest part of the small intestine. It is retroperitoneal except for the first part. It has the characteristic mucus secreting glands called Brunner glands.

The duodenum consists of four parts:

- ***First part:*** It extends from the pyloric orifice of the stomach to the neck of the gallbladder. Length of the first part is about 5cms. Most duodenal ulcers are found to occur in this part of the duodenum.

- ***Second part:*** Length of the second part is about 7.5cms. It lies just to the right of midline and extends from the neck of the gallbladder to the lower border of L3 vertebra.
- ***Third part:*** Length of the third part of duodenum is around 10cms. It is anterior to the inferior vena cava, the aorta, and the vertebral column. It lies posterior to superior mesenteric artery and vein.
- ***Fourth part:*** Length of fourth part of duodenum is around 2.5cms. It runs upwards and terminates at the duodenojejunal flexure.

2. JEJUNUM:

The length of jejunum and ileum together is around 4-6 metres. The jejunum constitutes the proximal two-fifths. It is larger in diameter and has a thicker wall than the ileum. Compared to ileum the arterial arcades are less prominent and the vasa recta are longer.

3. ILEUM:

The ileum makes up the distal three-fifths of the small intestine. Ileum has thinner walls, shorter vasa recta, more mesenteric fat, and more arterial arcades than jejunum. It has the narrowest lumen of the small bowel. Ileum terminates at the ileo-cecal junction which is guarded by the ileocecal valve internally. [2, 3]

LARGE INTESTINE:

The large intestine extends from the terminal end of the ileum to the anus. The length of large intestine is approximately 1.5 m. The parts of large intestine include cecum, appendix, ascending, transverse, descending and sigmoid colon, the rectum and the anal canal.

The cecum is the first part of the large intestine. It is a blind pouch that lies inferior to the ileocecal opening. It is intraperitoneal and is continuous with the ascending colon at the entrance of the ileum.

The appendix is a narrow, blind-ending tube that is connected to the cecum. Length of the appendix varies from 6-9cms. It walls contains large aggregations of lymphoid tissue. Appendix is suspended by the mesoappendix from the terminal ileum, which contains the appendicular vessels. The three taeniae coli merge into the base of the appendix.

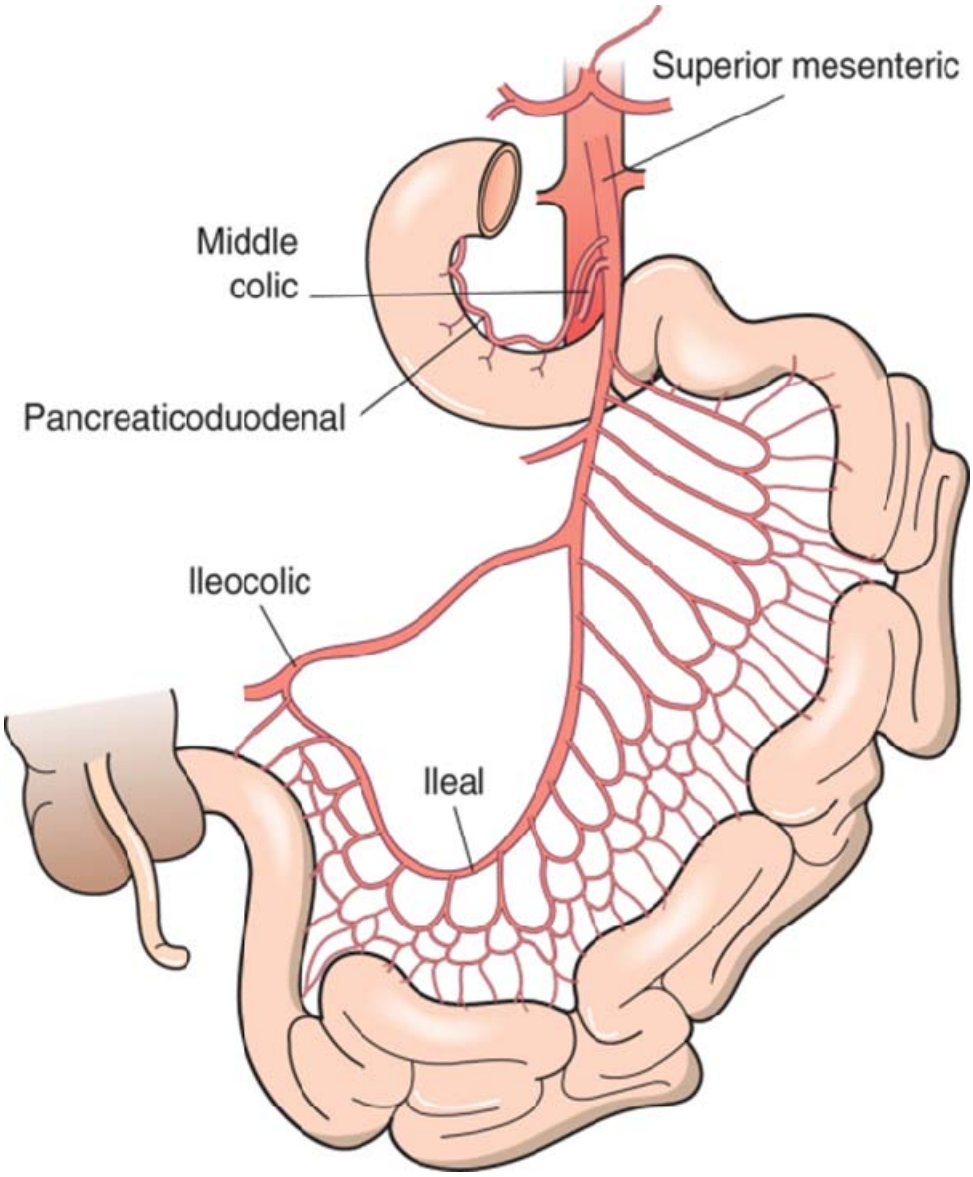
The colon extends superiorly from the cecum and consists of the ascending colon, transverse colon, descending colon, and sigmoid colon. The ascending and descending colon are retroperitoneal and the transverse and sigmoid colon are intraperitoneal. The right colic or hepatic flexure is at the junction of the ascending and transverse colon. Similarly left colic flexure or splenic flexure is at the junction of the transverse and descending colon. The final segment of the colon (the sigmoid colon) begins above the pelvic inlet and extends to the level of vertebra S3, where it is continuous with the rectum. Approximate length each segment is as follows, Ascending colon-15cms, transverse colon- 45cms, descending colon-30cms, and sigmoid less than 45cms.

Rectum and anal canal: Continuation of the sigmoid colon is the rectum. The recto sigmoid junction is usually at the level of S3 vertebra. The anal canal is the continuation of the large intestine after the rectum. Rectum is about 12cms in length and the anal canal is about 4cms in length. [2, 3, 4]

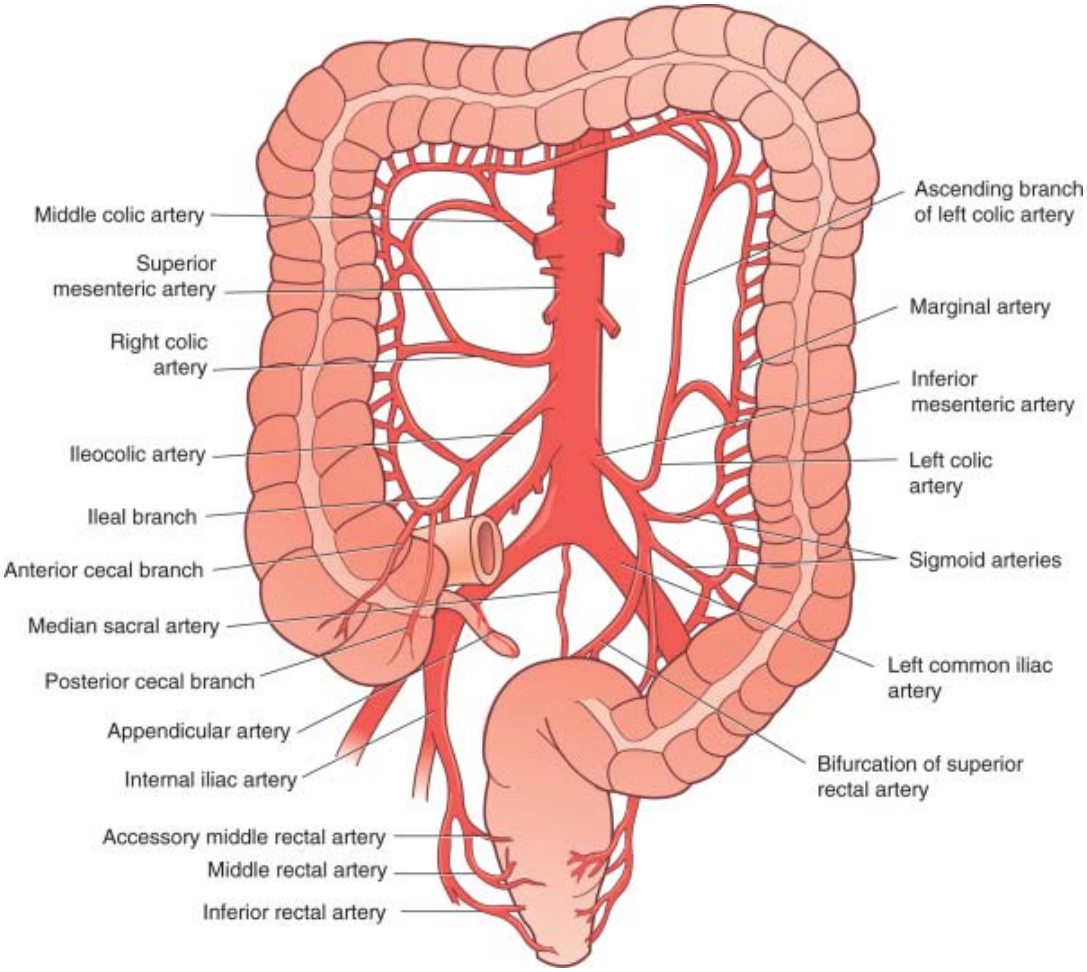
ARTERIAL SUPPLY:

- ✓ The celiac artery supplies the proximal half of the duodenum.
- ✓ The superior mesenteric artery supplies the rest of the duodenum, the jejunum, the ileum, the ascending colon, and the proximal 2/3rd of the transverse colon.
- ✓ The inferior mesenteric artery supplies the distal 3rd of the transverse colon, the descending colon, the sigmoid colon, and most of the rectum.
- ✓ The middle rectal artery supplies the lower part of the rectum.
- ✓ Part of the anal canal above the pectinate line is supplied by the superior rectal artery and the part below by inferior rectal artery.

ARTERIAL SUPPLY OF SMALL INTESTINE



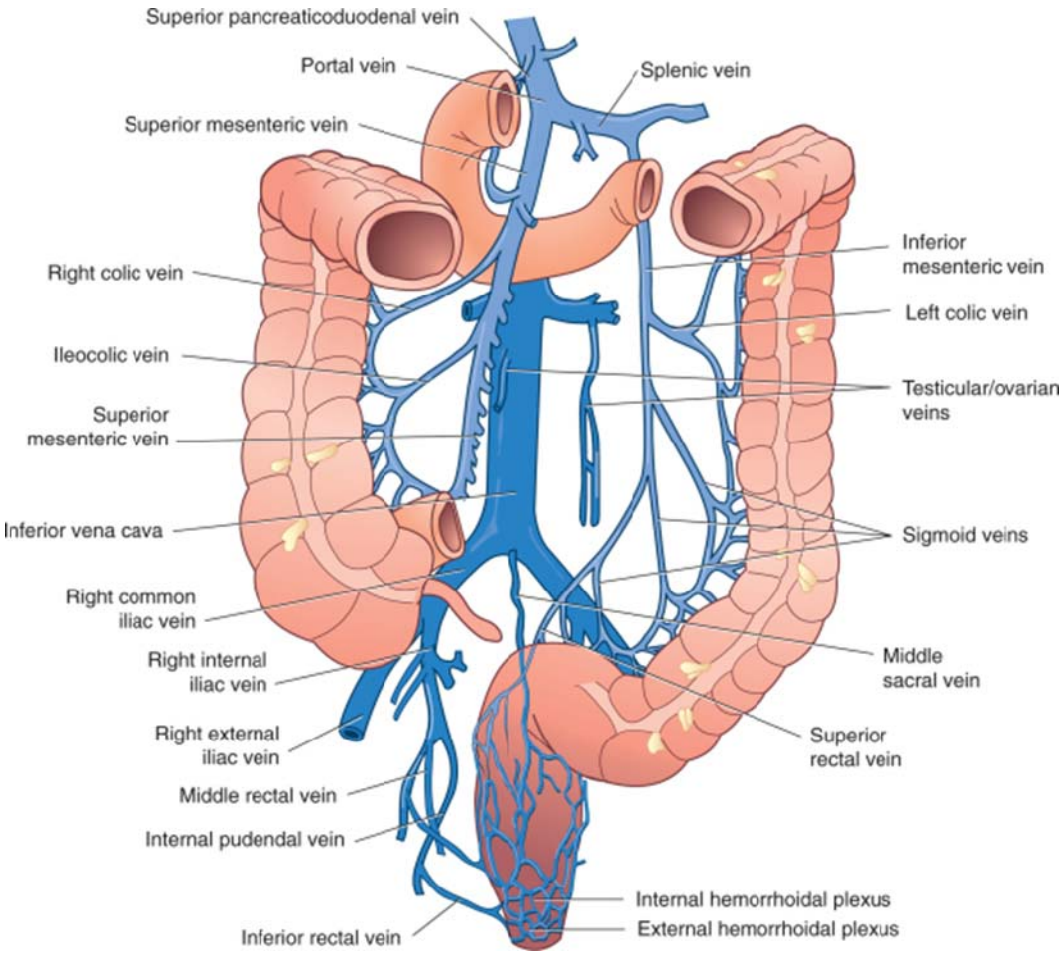
BLOOD SUPPLY OF LARGE INTESTINE



VENOUS DRAINAGE:

- ✓ Duodenum drains into splenic, superior mesenteric and portal veins.
- ✓ Small intestine, cecum, ascending colon, and transverse colon drain into the superior mesenteric vein. Behind the neck of the pancreas, the superior mesenteric vein joins the splenic vein to form the portal vein.
- ✓ The rectum, sigmoid colon, descending colon, and splenic flexure drain into the inferior mesenteric vein. It begins as the superior rectal vein and ascends, receiving tributaries from the sigmoid veins and the left colic vein.
- ✓ The lower two third of the rectum and anal canal drains into the middle and inferior rectal veins which drains into internal Iliac vein.

VENOUS DRAINAGE OF LARGE INTESTINE



LYMPHATIC DRAINAGE:

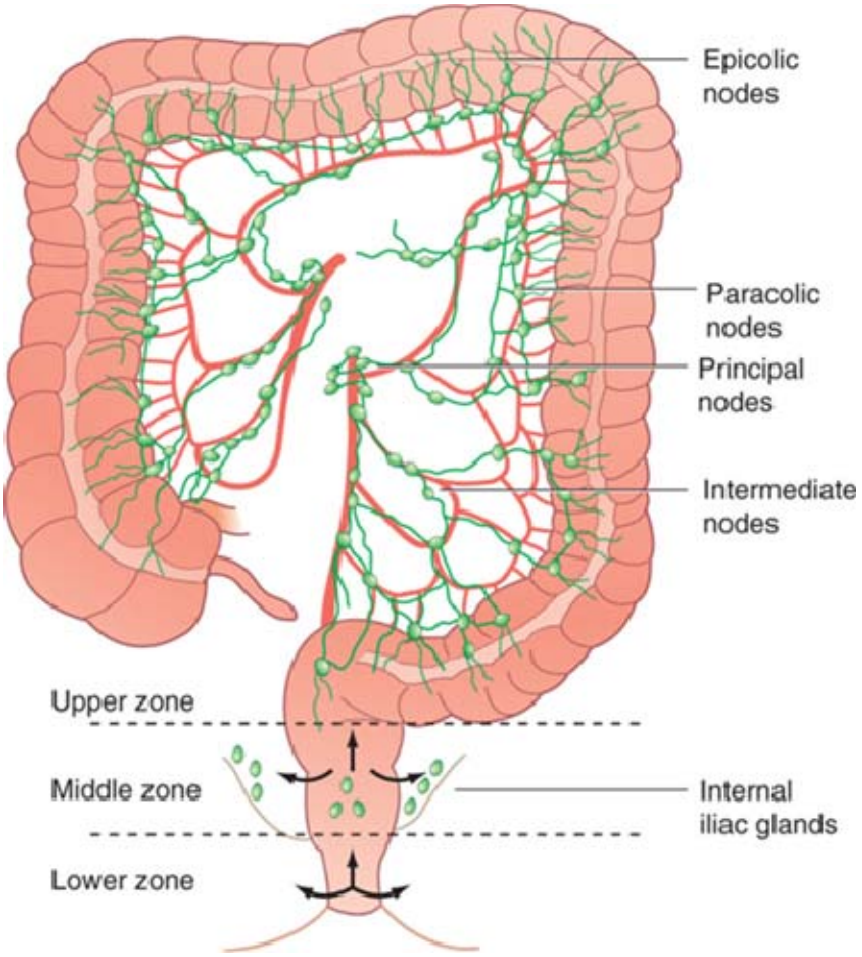
✓ Lymphatic drainage of **small intestine** occurs through lymphatic vessels coursing parallel to corresponding arteries. This lymph drains through mesenteric lymph nodes to the cisterna chyli, then through the thoracic duct, and ultimately into the left subclavian vein.

✓ The lymphatic drainage of the **colon** originates in a network of lymphatics in the muscularis mucosa. Lymph nodes that are found on the bowel wall are called as **epicolic nodes**. The nodes along the inner margin of the bowel close to the arterial arcades are called as **paracolic nodes**. The nodes around the named mesenteric vessels are called as **intermediate nodes** and the nodes at the origin of the superior and inferior mesenteric arteries are called as **main nodes.**

✓ Lymphatic drainage of the **rectum** parallels the vascular supply. The inferior mesenteric lymph nodes drain the upper and middle rectal lymphatic channels. The lower rectal lymphatic's drain laterally into the internal iliac lymph nodes and superiorly into the inferior mesenteric lymph nodes.

✓ The **anal canal** has a more complex pattern of lymphatic drainage. Proximal to the dentate line, lymph drains into both the inferior mesenteric lymph nodes and the internal iliac lymph nodes. Distal to the dentate line, lymph primarily drains into the inguinal lymph nodes, but can also drain into the inferior mesenteric lymph nodes and internal iliac lymph nodes.

LYMPHATIC DRAINAGE OF LARGE INTESTINE



NERVE SUPPLY:

✓ The parasympathetic of **the small intestine** is derived from the vagus and the sympathetic innervation of the small intestine is derived from the splanchnic nerves.

✓ **The colon** is innervated by both sympathetic and parasympathetic nerves. Sympathetic nerves (inhibitory) arise from T6-T12 and L1-L3. The parasympathetic (stimulatory) nerve supply to the right and transverse colon is from the vagus. The parasympathetic nerve supply to the left colon arises from sacral nerves S2-S4 to form the Nervi erigentes.

✓ Both sympathetic and parasympathetic nerves innervate the **anorectum.** Sympathetic nerve fibers are derived from L1-L3 and join the preaortic plexus. The preaortic nerve fibers then extend below the aorta to form the hypogastric plexus. Parasympathetic nerve fibers are known as the Nervi erigentes arising from S2-S4. These fibers join the sympathetic fibers to form the pelvic plexus.

✓ The **internal anal sphincter** is innervated by sympathetic and parasympathetic nerve fibers; both types of fibers inhibit sphincter contraction.

✓ The **external anal sphincter and puborectalis** muscles are innervated by the inferior rectal branch of the internal pudendal nerve. The levator ani receives innervation from both the internal pudendal nerve and direct branches of S3 to S5.

✓ **Sensory innervation to the anal canal is** provided by the inferior rectal branch of the pudendal nerve. While the rectum is relatively insensate, the anal canal below the dentate line is sensate. [5, 6]

CLASSIFICATION OF INTESTINAL OBSTRUCTION

Intestinal obstruction can be classified in the following ways,

1. Based on the site of obstruction as;
    a) Proximal Small bowel obstruction
    b) Distal small bowel obstruction
    c) Large bowel obstruction.
2. Based on the etiology as;
    a) Mechanical obstruction or
    b) Functional obstruction.
3. Based on the time of presentation as;
    a) Acute or
    b) Chronic intestinal obstruction.
4. Based on the extent of obstruction as;
    a) Partial or
    b) Complete obstruction.

5. Based on the type of obstruction as;
    a) Simple,
    b) Closed loop, or
    c) Strangulated obstruction.
6. Mechanical obstruction can be,
    a) Extra luminal
    b) Intramural
    c) Intra luminal.
7. Functional bowel obstruction can be due to,
    a) Intra-Abdominal or
    b) Extra-Abdominal causes.

ETIOLOGY:

The etiology of intestinal obstruction is either mechanical (dynamic) or functional obstruction (adynamic).

The causes of dynamic obstruction are the following,

I) Lesions outside the Intestinal Wall:

A) ADHESIONS

1. Postoperative
2. Congenital
3. Post inflammatory.

B) HERNIA

1. Internal Hernia includes Para duodenal, foramen of Winslow, diaphragmatic, mesenteric defect, paracecal, intersigmoid, broad ligament.
2. External Hernia, includes inguinal, femoral, umbilical, ventral, epigastric, lumbar, interstitial etc.
3. Incisional Hernia.

C) CONGENITAL

1. Malrotation
2. Annular pancreas
3. Omphalomesentric duct remnant.

D) NEOPLASTIC

1. Extra intestinal neoplasm
2. Carcinomatosis

E) INFLAMMATORY

1. Intra-abdominal abscess
2. "Starch" peritonitis.

F) MISCELLANEOUS

1. Volvulus
2. Gossypiboma
3. Superior mesenteric artery syndrome

II) Lesions in the Intestinal Wall:

A) CONGENITAL

1. Meckel's diverticulum
2. Intestinal atresia
3. Duplications/cysts

B) INFLAMMATORY

1. Crohn's disease
2. Eosinophilic granuloma

C) INFECTIONS

1. Tuberculosis
2. Actinomycosis

D) NEOPLASTIC

Primary/ Metastatic neoplasm’s

E) MISCELLANEOUS

1. Intussusception
2. Endometriosis
3. Radiation enteropathy
4. Ischemic stricture.

III) Lesions in the lumen:

1. Gallstone ileus
2. Round worm
3. Meconium ileus
4. Phytobezoar
5. Swallowed foreign body

The causes of functional bowel obstruction are the following,

I) Intra-Abdominal Causes

A) INTRAPERITONEAL PROBLEMS

1. Peritonitis
2. Intra-abdominal abscess
3. Postoperative
4. Autoimmune
5. Intestinal ischemia.

B) RETROPERITONEAL PROBLEMS

1. Metastasis
2. Urolithiasis/ Pyelonephritis
3. Pancreatitis
4. Retroperitoneal trauma/hematoma

II) Extra-Abdominal Causes

A) THORACIC PROBLEMS

1. Myocardial infarction
2. Congestive heart failure
3. Pneumonia
4. Thoracic trauma

B) METABOLIC ABNORMALITIES

1. Electrolyte imbalance
2. Sepsis
3. Lead poisoning
4. Porphyria
5. Ketoacidosis
6. Hypothyroidism
7. Hypoparathyroidism
8. Uremia

C) MEDICINES

1. Anticholinergics
2. Antihistamines
3. Opiates
4. Alpha-adrenergic agonists
5. Catecholamine’s

D) MISCELLANEOUS

1. Spinal cord injury
2. Pelvic fracture
3. Head trauma
4. Chemotherapy
5. Radiation therapy
6. Renal transplantation

Among these post-operative adhesions, hernias and neoplasms accounts for most of the cases. [7, 8, 9, 10]

ADHESIONS:

✓ Adhesions are defined as abnormal connective tissue attachments between tissue surfaces. They can be classified as

1. Congenital adhesions or
2. Acquired (post inflammatory and postoperative).

✓ Postoperative adhesions are the most common cause of small bowel obstruction in Western countries. They account for 40–80% of cases of acute intestinal obstruction.

✓ It is caused by the activation of the complement and coagulation cascades, along with exudation of fibrinogen rich fluid; the full establishment of this response is present 5–7 days after the peritoneal trauma.

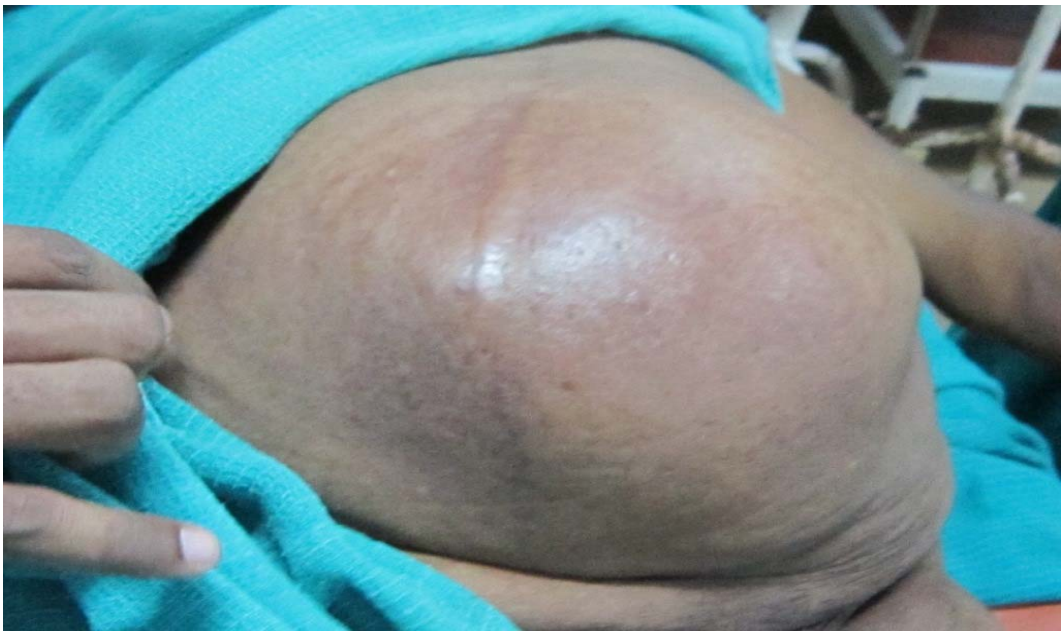
✓ Adhesions occur due to the formation of fibrin gel matrix which consists of numerous types of cells, such as leukocytes, platelets, mast cells, and erythrocytes, as well as surgical debris and also bacteria.

- ✓ It is adynamic process. In the initial phase macrophages predominate, by 2-4 days fibrin and fibroblasts appear and by 5^{th} day fibroblasts begin to form syncytium.
- ✓ The type of operation is an important factor. The operations most frequently associated with adhesion are those involving the structures in the inframesocolic compartment.
- ✓ Adhesive bowel obstruction may occur at any time ranging from as early as within the first postoperative month to more than 8 decades after the index operation. [7, 11, 14, 15, 16, 17]

HERNIA:

- ✓ Hernias are the second most common cause of intestinal obstruction world wide after adhesive obstruction Hernias are more common in males than in females, since inguinal hernias are more common in men.
- ✓ External hernias if not operated electively will go for obstruction in the future requiring an emergency surgery.

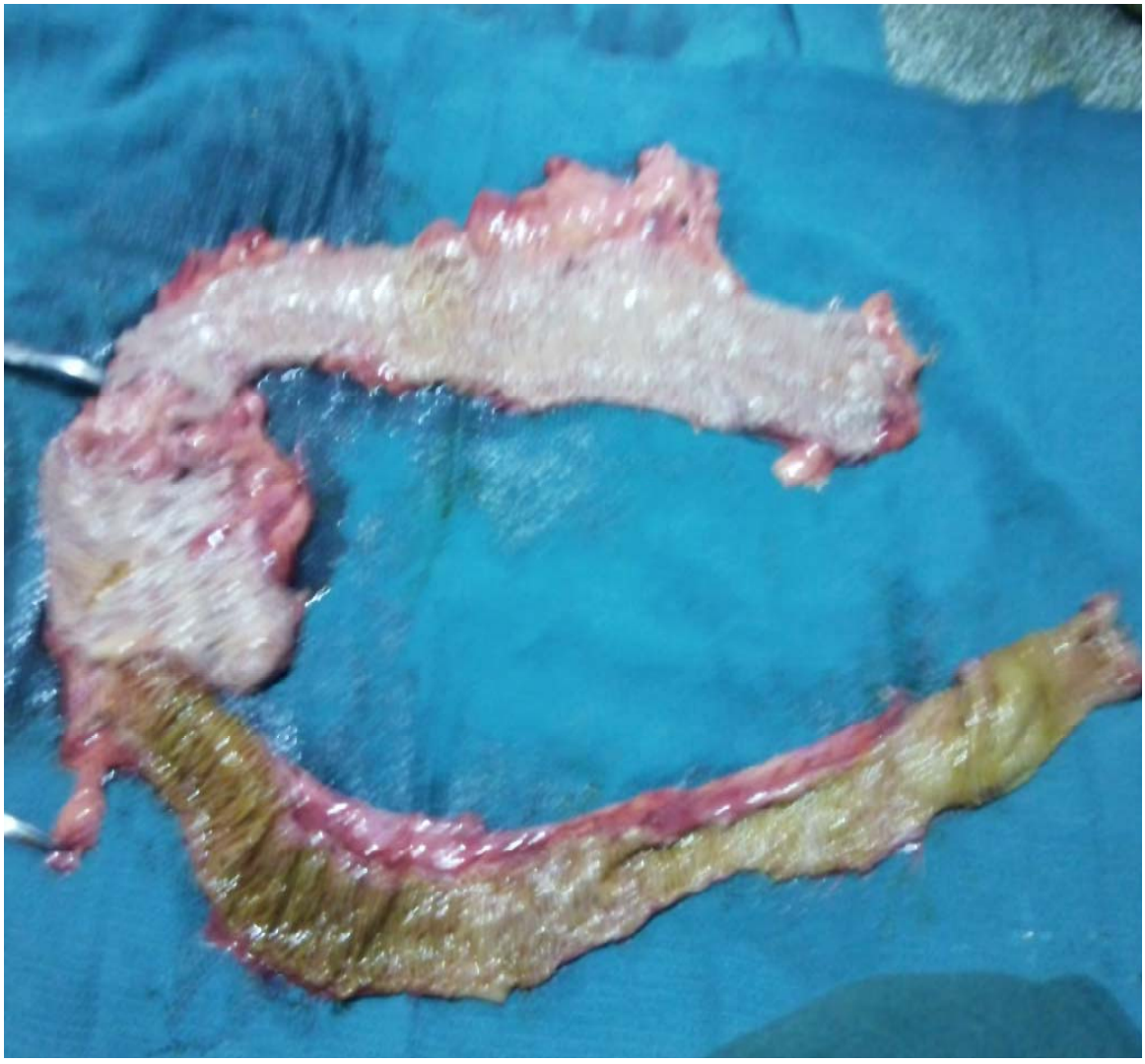
OBSTRUCTED INGUINAL HERNIA



OBSTRUCTED INCISIONAL HERNIA

MALIGNANT BOWEL OBSTRUCTION:

- ✓ Patients with malignant obstruction are usually elderly with comorbidities that affect the outcome of surgery.
- ✓ Malignant bowel obstruction can be either due to primary neoplasms or due to metastatic cancer.
- ✓ Primary neoplasm's arising from Colon, rectum, stomach; small bowel and ovary are the common causes of malignant bowel obstruction.
- ✓ Metastatic cancer can also cause bowel obstruction. The most common metastatic cancer causing obstruction is peritoneal carcinomatosis, but melanoma and carcinoma of the breast, kidney, or lung can also cause obstruction due to metastasis. [7, 11, 18]



ASCENDING COLON GROWTH CAUSING

OBSTRUCTION

GRANULOMATOUS DISEASES:

- ✓ Among the granulomatous diseases Tuberculosis accounts for most of the cases in India.
- ✓ Other granulomatous diseases causing obstruction are Crohns and Actinomycosis.

INTUSSUSCEPTION:

- ✓ It is the telescoping or invagination of one segment of bowel into the adjacent segment.
- ✓ It can be either antegrade or retrograde.
- ✓ It is frequent cause of obstruction during the first 2 years of life but in adult population it is very rare.
- ✓ Etiology of Intussusception is idiopathic in children's and due to inflammation and edema of payer's patches, but in adults it occurs due to submucus lipoma, leiomyoma, jejunal polyps, other polyps and carcinomas with papillary projections.

VOLVULUS:

- ✓ Volvulus is the axial twist of the bowel and its mesentery either clockwise or anticlockwise.
- ✓ About 15 % of the large bowel obstruction is due to volvulus.
- ✓ Sigmoid volvulus is the most common type accounting for 65% of patients with volvulus. The rotation is usually anti-clockwise. Caecum is the 2nd common site.
- ✓ It is common in Ogilvie's syndrome, mentally retarded, Chaga's disease, Hypothyroidism, Anti-cholinergic drugs, Multiple sclerosis, Scleroderma and Parkinson's disease.

OTHER CAUSES:

Other causes of acute intestinal obstruction are congenital lesions, infections, inflammation, meconium pellets, stricture, gallstones, foreign body, parasitic infestation, post traumatic lesions and miscellaneous conditions. [7]

SIGMOID VOLVULUS

SIGMOID VOLVULUS WITH GANGRENE

PATHO PHYSIOLOGY

Intestinal obstruction results in prominent alterations of the normal intestinal physiology. **Bowel distension, decreased absorption, intraluminal hypersecretion, and alterations in motility** are found universally.

In the past it was thought that the decrease in blood flow to intestinal wall was responsible for most of the pathophysiologic changes. But recent studies suggests that many of the pathophysiologic changes that occurs during intestinal obstruction are due to the increases in blood flow seen during the early phases of bowel obstruction due to intramural inflammation. Mucosal production of reactive oxygen species plays a vital role. [12]

Pathophysiological changes of acute intestinal obstruction can be studied under the following headings:

1. Intestinal distention
2. Intestinal motility
3. Circulatory changes and
4. Microbiological changes

Intestinal Distention:

Bowel distension is a constant feature of bowel obstruction. Most of the gas distending the small bowel in the early phases of obstruction accumulates from swallowed air. Nitrogen constitutes 75% of luminal gas.

Other sources include:

✓ Fermentation of sugars,

✓ Production of carbon dioxide by interaction of gastric acid and bicarbonates in pancreatic and biliary secretions, and

✓ Diffusion of oxygen and carbon dioxide from the blood.

The nitric oxide produced due to inflammation causes smooth muscle relaxation, further aggravating the distention. The reactive oxygen species generated not only affect gut motility but also affects its permeability.

Two phases of obstruction were noted,

- ✓ During the first 12 hours of an obstruction of the small bowel, water and electrolytes accumulate within the lumen due to decrease in net absorption.
- ✓ By 24 hours, intraluminal water and electrolytes accumulate more rapidly secondary to a further decrease in absorption and in addition to an increase in net intestinal secretion secondary to mucosal injury and increased permeability.

The decrease in the absorptive capacity and increase in secretion leads to dehydration. Although the intestinal wall distal to the obstruction maintains relatively normal function, the inability of the luminal content to reach the unobstructed gut further aggravates dehydration.

Intestinal Motility:

In the early phase of bowel obstruction, intestinal contractile activity increases in an attempt to propel intraluminal contents past the obstruction. Later it diminishes due to intestinal wall hypoxia and the exaggerated intramural inflammation. [13]

Some investigators have suggested that the alterations in intestinal motility are secondary to a disruption of the normal autonomic parasympathetic (vagal) and sympathetic splanchnic innervations.

It is also proposed that loss of functions of interstitial cells of Cajal during intestinal obstruction affect the gut motility.

Circulatory changes:

Ischemia of the bowel wall can occur by the following mechanisms:

- ✓ Extrinsic compression of the mesenteric arcades by adhesions, fibrosis, a mass, or a hernia defect;
- ✓ Axial twist of the mesentery;
- ✓ Extrinsic pressure on bowel wall (e.g., a fibrous band); or
- ✓ Progressive distention due to closed-loop bowel obstruction.

The consequences of vascular compromise are more disastrous in large bowel, due to competent ileocecal valve, which functionally leads to a form of closed-loop obstruction. Progressive distention of the bowel lumen along with increase in the intraluminal pressure results in increased transmural pressure on capillary blood flow within the wall of the bowel. Venous obstruction occurs first followed by arterial occlusion; leading to ischemia. It is most commonly seen in the ascending colon.

Microbiology:

The upper small intestine contains gram-positive facultative organisms in small concentrations, usually $<10^6$ colonies/ml.

More distally, the bacterial count increases in concentration to about 10^8 colonies/ml in the distal ileum, and the flora changes to primarily coliforms and anaerobic organisms.

In the presence of obstruction,

✓ A rapid proliferation of bacterial organisms occurs proximal to the point of obstruction, in direct proportion to duration of obstruction, reaching a plateau of 10^9–10^{10} colonies/ml after 12–48 hours of an established obstruction.

✓ The bowel distal to the obstruction tends to maintain its usual bacterial flora. Due to the above said changes the bacterial flora during acute intestinal obstruction, the risk of infective complications are very high.

CLINICAL FEATURES

SYMPTOMS:

The classic clinical picture of a patient suffering from bowel obstruction includes crampy abdominal pain, vomiting, distention, and absolute constipation.

Abdominal pain:

Pain is the first symptom to develop which is acute in onset. Initial colicky pain suggests obstruction and eventual diffuse persistent pain suggests strangulation. Pain begins usually around the umbilicus in small bowel obstruction.

- ✓ In small bowel obstruction, pain is crampy, recurrent paroxysms occurring as short crescendo/decrescendo episodes of 30 seconds.
- ✓ In large bowel obstruction, it occurs as episodes of minutes.
- ✓ In paralytic ileus, pain is diffuse and mild.

Vomiting:

The more proximal the obstruction, the earlier and more prominent are the symptoms of nausea and vomiting. In large bowel obstruction, vomiting is a late feature.

Initially it contains food particles and later it becomes bilious. Finally with complete obstructions and bacterial proliferation it becomes feculent.

Distension:

It is absent or minimal in jejunal obstruction. In terminal ileal obstruction it is obviously seen with visible intestinal peristalsis and borborygmi sounds. It is enormous in large bowel obstruction.

Obstipation:

Obstipation means failure to pass both feces and flatus. It occurs earlier in large bowel obstruction.

PAST HISTORY:

The past medical history of the patient may be key in making both the diagnosis and establishing the cause. It is especially important to inquire about previous events of bowel obstruction, recent and distant abdominal operations, current medications, a history of chronic constipation, recent changes in the caliber of stools, a history of cancer and its stage at presentation and related treatments (surgery, chemotherapy, or radiation therapy), and a history of Crohn's disease.

PHYSICAL EXAMINATION:

A thorough physical examination is mandatory and should include assessment of vital signs and hydration status, abdominal inspection, palpation, auscultation, a search for potential hernia defects, and a rectal examination.

Dehydration:

Tachycardia, hypotension, and oliguria are signs of dehydration. It should be treated aggressively; otherwise it will result in renal failure.

Temperature:

Fever may be associated with an inflammation in the bowel wall/ ischemia / perforation. Hypothermia occurs when septicemia develops, which is a poor prognostic sign.

Inspection:

On inspection one should look closely for abdominal distention and previous surgical incisions; including inguinal incisions to rule out recurrent hernias.

Palpation:

Abdominal palpation will reveal the presence of peritoneal signs such as localized tenderness, rebound tenderness, guarding and rigidity. One should also look for abdominal masses.

Auscultation:

Bowel sounds are increased in mechanical bowel obstruction initially, characteristically heard as high-pitched metallic "rushes" and "groans" followed by the metallic tinkling sounds. Eventually once fatigue occurs or gangrene develops, bowel sounds are not heard. In functional bowel obstruction only metallic tinkling is heard indicative of dilated bowel. [7, 8]

INVESTIGATIONS:

Laboratory:

Laboratory data, although nondiagnostic, may be helpful in determining the condition of the patient and guides the resuscitation.

- ✓ A complete blood cell count and differential, electrolyte panel, blood urea nitrogen, creatinine, and urinalysis should be obtained to evaluate fluid and electrolyte imbalance and to rule out sepsis.

- ✓ Arterial blood pH, serum lactate, and amylase and lactic dehydrogenase activity are useful tests in the evaluation of bowel obstruction, especially to rule out bowel necrosis.
- ✓ Others have suggested that serum concentrations of phosphate, intestinal fatty acid binding protein, and isoforms of creatine phosphokinase (isoform B) may identify the presence of intestinal cell necrosis; however, the specificity and the sensitivity are not accurate.

Radiological studies:

Chest and Abdominal Radiographs:

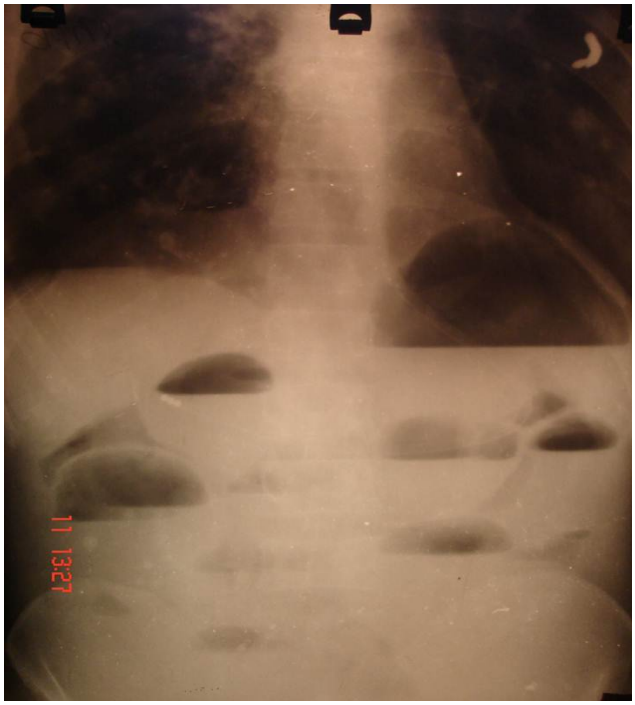
An upright chest x-ray combined with supine and upright abdominal radiographs should be done in all cases. The chest x-ray is helpful to detect extra-abdominal conditions that may present with a clinical picture similar to bowel obstruction (e.g., pneumonia) and the presence of sub diaphragmatic free air indicative of a perforated viscus.

Plain X-ray abdomen erect view will show the following features;

- ✓ Multiple air-fluid levels indicate intestinal obstruction.
- ✓ Proximal bowel obstruction has lesser air-fluid levels.
- ✓ Distal bowel obstruction usually has more number of air-fluid levels.
- ✓ Normally, three air-fluid levels are seen – one at the fundus, second at duodenum and third at caecum.
- ✓ The small intestine is considered dilated if loops of bowel measure more than 3 cm in diameter, similarly proximal large bowel >9cms, transverse colon >5.5cms, sigmoid colon >5cms are considered to be dilated.
- ✓ Jejunum shows concertina effect due to volvulae conniventes (herring bone pattern)
- ✓ Ileum is smooth and characterless
- ✓ Large bowel shows haustrations.
- ✓ Sigmoid volvulus shows bent inner tube appearance or coffee bean appearance.

Differentiation between small and large intestine can be made by the following ways,

- ✓ Dilated loops of small intestine tend to lie in the central portion of the abdomen and are recognized by the presence of the valvulae conniventes or plicae circulares that traverse the full diameter of the bowel.
- ✓ Dilated segments of large intestine are usually visualized in the periphery of abdominal films and are identified by the presence of haustral markings that only partially traverse the bowel wall. [7, 8]



X- ABDOMEN ERECT VIEW- MULTIPLE AIR FLUID LEVELS

COFFEE BEAN APPEARANCE IN SIGMOID VOLVULUS

Contrast Studies:

The use of contrast is helpful when the diagnosis is uncertain in patients with a nonresolving partial small bowel obstruction and to differentiate between partial and complete bowel obstruction. It can also identify the specific site and often the cause of the obstruction.

a) Antegrade approach:

Contrast is given orally or through Ryle's tube. Barium swallow, Barium meal and Barium meal follow through comes under this.

b) Retrograde contrast study (barium enema):

It can be useful in the patient with suspected large bowel obstruction or in those with a clinical and radiologic picture of a distal small bowel obstruction but with no history of abdominal operations or evidence of an external hernia.

Contrast agent of choice:

Commonly used agents are,

1. Dilute barium- it allows a better visualization of mucosal detail and
2. Water-soluble contrast.

The use of both types of contrast agent has risks.

- ✓ In patients with large bowel obstruction, barium within the lumen of the colon proximal to the site of obstruction can become inspissated and itself cause a complete obstruction. Also intraperitoneal extravasation of the luminal barium contrast will cause infective complications and barium peritonitis.
- ✓ Gastrografin and other water-soluble agents are relatively safe even if extravasation occurs, but other potential complications have been reported. Pulmonary aspiration of these agents is more troublesome than that of barium. [19, 20, 21]

Computed Tomography:

Computed tomography (CT) has become a valuable tool in the diagnosis of intestinal obstruction, particularly when strangulation is suspected. CT will give an idea about both the site as well as the etiology of the obstruction. It has got a sensitivity of 93%, specificity of up to 100%, and accuracy of 94% in diagnosing intestinal obstruction.

CT findings diagnostic of bowel obstruction include

- ✓ Intestinal loops greater than 25 mm in diameter and
- ✓ A transition zone between dilated and collapsed bowel loops.

Another advantage of CT is its ability to visualize the entire intra-abdominal compartment as well as defects in the abdominal wall. In addition CT can demonstrate changes in the intestinal wall and associated mesentery.

Ultrasonography:

It is useful to see dilated bowel and fluid in the peritoneal cavity. It is better than X-rays but not as good as CT scan. Doppler ultrasound is useful in detecting strangulation. [22, 23]

MANAGEMENT:

A) SMALL BOWEL OBSTRUCTION:

Initial management:

In patients with small bowel obstruction, the initial management includes

- Fluid resuscitation,
- Naso-gastric decompression and prevention of aspiration.
- Blood sample for serum electrolyte concentrations, cross-matching and arterial blood gas analysis.
- Metabolic or electrolyte imbalances should be corrected.

The above said steps are the same for all patients, whether they are managed operatively or nonoperatively.

Fluid resuscitation:

The most important initial step in management is vigorous fluid resuscitation.

- Patients with small bowel obstruction often present with profound volume losses and may require large amounts of isotonic crystalloids such as normal saline or Ringer's lactate.
- Resuscitation should be guided by urine output, provided the patient is hemodynamically stable with normal renal function.
- Patients who are hemodynamically unstable may require hemodynamic monitoring of central venous or pulmonary arterial pressure to evaluate their volume status.

Nasogastric decompression:

Apart from reducing the intestinal distention from swallowed air, Nasogastric decompression also prevents aspiration. It can improve ventilation in patients with respiratory compromise. [24, 25]

Nonoperative Management:

A trial of nonoperative management should be considered only in patients with uncomplicated small bowel obstruction. The success rate is 62–85% in patients treated by this approach.

The success rate depends on,

- Type of bowel obstruction (complete / partial, or recurrent),
- Patient selection,
- Etiology (e.g., adhesions, hernia, or neoplasm),
- The surgeon's threshold for conversion to operative management.

Advantages:

- Patients successfully managed nonoperatively require shorter hospital stays and
- Do not experience the morbidity or convalescence necessitated by an operation.

Disadvantage:

- Recurrence rates will be greater with nonoperative management.

Contraindications:

Contraindications to nonoperative management include

- Suspected ischemia,
- Large bowel obstruction,
- Closed-loop obstruction,
- Strangulated hernia, and
- Perforation.
- A relative contraindication to nonoperative management is complete small bowel obstruction.

Nonoperative treatment principles:

When patients with a small bowel obstruction are initially managed nonoperatively, following treatment principles needs to be considered.

- Adequate proximal decompression by Nasogastric tube.

- Patients managed nonoperatively should be aggressively resuscitated with crystalloids to prevent dehydration.
- Electrolytes should be monitored frequently and corrected. [26, 27, 28, 29]

When to Convert to Operative Management:

If a patient being treated nonoperatively develops evidence of a complicated obstruction, operative intervention is indicated.

Signs and symptoms suggestive of a complicated obstruction include

- Fever,
- Tachycardia,
- Leukocytosis,
- Localized tenderness,
- Abdominal pain,
- Peritonitis.

The presence of any four of the above signs indicates strangulation. Patients who develop free air or signs of a closed-loop obstruction on abdominal radiograph require operative exploration.

If evidence of ischemia, strangulation, or vascular compromise is noted on CT, such as pneumatosis intestinalis, bowel wall thickening, portal venous gas, generalized ascites, or nonenhancement of the bowel wall, then operative intervention is usually indicated.

The timing of conversion to operative management in a patient with a small bowel obstruction who is not improving with nonoperative management is controversial. Some surgeons advocate surgical intervention in any patient who fails to show improvement within 48 hours of initiating therapy. Others advocate waiting up to 4-6 days.

OPERATIVE MANAGEMENT:

Once the decision has been made to pursue operative management, steps should be taken to prevent peri- and postoperative complications.

Preoperative preparation includes

- Assessing and addressing the medical fitness of the patient,
- Taking steps to optimize the patient's medical status.
- Consideration should be given to the administration of beta-blockers to patients with cardiovascular comorbidities.
- Special consideration should be given to ensuring that the patient has been resuscitated adequately, appropriate antibiotics have been given, and any electrolyte abnormalities have been addressed.
- A nasogastric tube should already be in place to decrease the risk of aspiration during the induction of anesthesia.

Several decisions must be made with regard to operative planning to provide the safest operation that will afford the best outcome for each individual patient.

The choice of operative approach and incision is important to allow the surgeon adequate exposure and visibility.

- A laparoscopic approach should at least be considered in patients with uncomplicated small bowel obstruction. Laparoscopy is known to cause fewer adhesions.
- When an obstruction develops in the early postoperative period, the original incision should be reopened.
- In patients without a history of operation or in those remote from their original operation, a midline incision affords the best exposure to all four quadrants of the abdomen.

Once within the abdominal cavity, the first step is to identify the site and cause of obstruction. If the point of obstruction is not obvious, decompressed bowel distal to the obstruction can be identified and followed proximally to the point of obstruction. The dilated bowel proximal to the offending obstruction is often very thin-walled and at increased risk for perforation.

After the offending obstruction has been corrected, a thorough exploration of all four quadrants should always be done. Nonviable bowel needs to be identified and resected. When a small bowel resection is necessary, intestinal continuity of the small bowel can generally be accomplished with a primary anastomosis unless there is generalized peritonitis. In patients with malignant small bowel obstruction or if the offending obstruction is unable to be released or it is deemed unsafe to attempt to dissect out the point of obstruction, intestinal bypass can be performed. Abdominal closure may be difficult to achieve when the small bowel is massively dilated. In these cases, intraoperative intestinal decompression will facilitate closure.

Techniques described for intraoperative decompression include

- Manual retrograde decompression into the stomach. This maneuver is the safest and quickest technique.
- Passage of a long nasointestinal tube, and
- Performance of a controlled enterotomy (discouraged). [30]

INTRA-OPERATIVE PICTURE OF OBSTRUCTED

INGUINAL HERNIA

INTRA-OPERATIVE PICTURE OF OBSTRUCTED

PARAUMBLICAL HERNIA

Recurrent Small Bowel Obstruction:

Recurrent obstruction is more common in patients with

- ✓ Multiple adhesions, matted adhesions,
- ✓ Previous admissions for small bowel obstruction, and
- ✓ Previous pelvic, colonic, and rectal surgery.

In the past, numerous attempts have been made by surgeons to control the formation of adhesions in an effort to prevent future mechanical obstruction.

1. A simple technique to prevent adherence of the bowel to the undersurface of the fascial incision is to interpose the omentum between the bowel and the incision.
2. The Noble plication and the Childs-Phillips transmesenteric plication. These procedures involve the suturing of adjacent loops of small bowel into an orderly pattern in an attempt to permanently plicate the bowel in a position that will not allow mechanical obstruction.

3. Another method used to prevent mechanical obstruction of the small bowel is intraluminal "stenting" or "splinting" of the bowel with a long intestinal tube.

Patients having had multiple previous obstructions are less likely to develop bowel ischemia related to adhesions because of the relative lack of mobility of the bowel. For these reasons it may be advisable to attempt nonoperative therapy a bit more liberally in patients with multiple recurrences.

Adhesion Prevention:

Multiple approaches have been employed to prevent the formation of postoperative adhesions.

- ✓ Washing peritoneal cavity with saline,
- ✓ Leaving raw peritoneal areas unsutured,
- ✓ Covering raw peritoneal surface with greater omentum,
- ✓ The formation of fibrin bridges (and thus adhesions) may be preventable by application of an absorbable "biofilm."

Malignant Obstruction:

Bowel obstruction in the setting of carcinomatosis often represents the terminal phase of the malignant disease. Surgical management is purely palliative and needs to be applied selectively.

B) LARGE BOWEL OBSTRUCTION:

Virtually all patients with complete acute large bowel obstruction require prompt surgical intervention and should not undergo a trial of nonoperative management. Acute complete large bowel obstruction in a patient with a competent ileocecal valve is a true surgical emergency because of the high risk of perforation. Once the diagnosis has been made, surgical exploration should be undertaken as soon as possible after appropriate resuscitation.

Prior to exploration, the same principles apply for large bowel obstruction as for small bowel obstruction which includes,

✓ Volume losses may be substantial, and patients should be resuscitated aggressively with an isotonic crystalloid solution.

- ✓ Electrolyte and acid-base abnormalities should be corrected.
- ✓ Nasogastric decompression is also important in patients with a large bowel obstruction to decrease the amount of air and gastric contents delivered to the bowel.
- ✓ A bladder catheter should be inserted to help guide resuscitation and as preparation for the operating room.
- ✓ As with small bowel obstruction, invasive monitoring may be necessary depending on the hemodynamic status of the patient.
- ✓ Antibiotics targeted at both skin and colonic flora should be administered.
- ✓ Possible stoma site should be before being taken to the operating room to help ensure appropriate placement of the stoma device.

Exploration in patients with large bowel obstructions is best performed through a low midline incision. Patients with large bowel obstructions should be placed in the lithotomy or modified lithotomy position if access to the anus is anticipated.

Once inside the abdominal cavity, the surgeon should proceed with exploration of all four quadrants as well as examination of the liver, omentum, and retroperitoneal lymph nodes for any suspicious lesions.

- ✓ Obstructing lesions of the cecum and ascending colon should be resected via right hemicolectomy, usually with a primary anastomosis.
- ✓ Lesions in the transverse colon should be managed with an extended right hemicolectomy and again, with a primary anastomosis.
- ✓ Proximal diversion with an end ileostomy is not necessary in all patients; however, proximal diversion should be considered when there is any concern about bowel viability, if the patient is unstable, or in the case of substantial peritoneal contamination or peritonitis.

The management of obstructing lesions in the descending, sigmoid colon and rectum is more controversial.

1. Intraoperative bowel preparation allows for segmental resection and primary anastomosis of the involved colon provided the remnant bowel to be reanastomosed is healthy and neither too edematous nor too dilated; this approach has an acceptable leak rate of around 5%.
2. Primary anastomosis should not, however, be carried out in the setting of fecal contamination, peritonitis, hemodynamic instability, or possible ischemia of the remaining colonic segments. Many surgeons prefer a more classic approach with a Hartmann's procedure of segmental resection of the affected colon, an end colostomy, and a blind distal pouch or mucous fistula.
3. A diverting loop ileostomy can be added to a primary anastomosis if there is any question of anastomotic integrity.

4. Another option for left-sided lesions is subtotal colectomy and primary ileosigmoidostomy or ileorectostomy. This approach may be advisable when there is concern about possible cecal perforation, when there are multiple serosal tears in the colon or if the patient had a previous colonic cancer and the current obstruction is from a new colonic carcinoma.

5. In ca rectum, if the patient is not a candidate for restoration of intestinal continuity, primary resection with end colostomy may be an acceptable approach.

6. Proximal colonic diversion can be accomplished, preferably using a diverting sigmoid or transverse loop colostomy. After discharge, patients with localized disease can undergo neoadjuvant therapy with definitive surgical resection after completing the chemo radiation. [31]

Laparoscopy:

Consideration should be given to an initial laparoscopic exploration in patients with obstructing or near obstructing lesions of the large bowel, and especially if proximal diversion alone is anticipated. Laparoscopic exploration has several advantages over exploratory laparotomy for obstructing colonic lesions, including shorter hospital stays, early return of bowel function, earlier initiation of neoadjuvant therapy, and the potential to provide a diagnosis when the cause of obstruction is not known preoperatively. [32]

SEMS:

Another option to consider in the early management of the patient with an obstructing lesion in the large bowel is the use of a self-expanding intraluminal metal stent (SEMS) to allow immediate colonic decompression and the ability to perform elective mechanical bowel preparation.

The use of SEMS is becoming widely available and it can be a useful tool for the surgeon managing a large bowel obstruction. In experienced hands, a SEMS can be placed successfully in about 90% of patients with low complication rates.

A SEMS can avoid the need for urgent or emergent operation by intraluminally decompressing the distended proximal colon and allowing distal passage of stool. This approach converts an otherwise emergent operation into an elective operation.

A SEMS is also useful when palliating patients who might not tolerate surgical diversion or those with unresectable disease and a limited survival. It can decrease the morbidity and mortality associated with an emergent operation, as well as increasing the rate of an eventual one-stage operation with primary anastomosis.

With a locally advanced obstructing rectal cancer, after placement of a SEMS, the patient can undergo neoadjuvant therapy followed by surgical resection, again increasing the chances for a successful one-stage operation. [33]

Ileus:

Postoperative ileus is usually differentiated from early small bowel obstruction by the presence of dilated small and large bowel. Currently, the treatment of ileus involves supportive measures only.

Nasogastric decompression is important to prevent further intestinal distension from swallowed air and secretions as well as to decrease the risk of vomiting and subsequent aspiration. Patients with an ileus can sequester a substantial volume in the dilated bowel, causing intravascular volume depletion; therefore these patients require diligent fluid and electrolyte replacement. Most importantly, the underlying cause of ileus needs to be addressed. Aggressive treatment of sepsis, electrolyte abnormalities, and associated intra-abdominal processes will hasten the return of bowel function. Ileus is almost always self-resolving and results in very few long-term sequelae.

Early postoperative ileus (in the first 3–5 days) after abdominal operation has been called a "physiologic" ileus by some. It is well known that the stomach, small bowel, and large bowel recover normal motor activity at different rates after operative trauma and anesthesia.

Within a few hours of operation, the small bowel returns to normal motor activity. The stomach recovers normal motor activity and emptying characteristics in 24–48 hours, while the large bowel takes 3–5 days to recover coordinated propulsive function.

In contrast to the more universal early postoperative ileus which is of limited duration, a generalized, adynamic ileus is much less common but may persist for many days to weeks in some patients.

Most investigators believe this motor problem to be a systemic disorder involving a dysregulation of neuromotor coordination. Every attempt should be made to correct electrolyte disorders, treat sepsis, and exclude a mechanical cause of potential obstruction.

Despite addressing all correctable abnormalities, on occasion the ileus can persist and can be a very vexing problem for both patient and physician, requiring prolonged gastric decompression and intravenous nutritional support.

Studies of pharmacological parasympathetic stimulation with parenteral neostigmine (on occasion with a concomitant sympatholytic agent) have been successful. This treatment should be given in a monitored setting.

PATIENTS AND METHODS

This study was conducted at Government Rajaji Medical College and Hospital for a period of two years from 2010 to 2012. It is an analytical study that included 100 patients who were diagnosed to have Acute Intestinal Obstruction based on clinical, biochemical and radiological features.

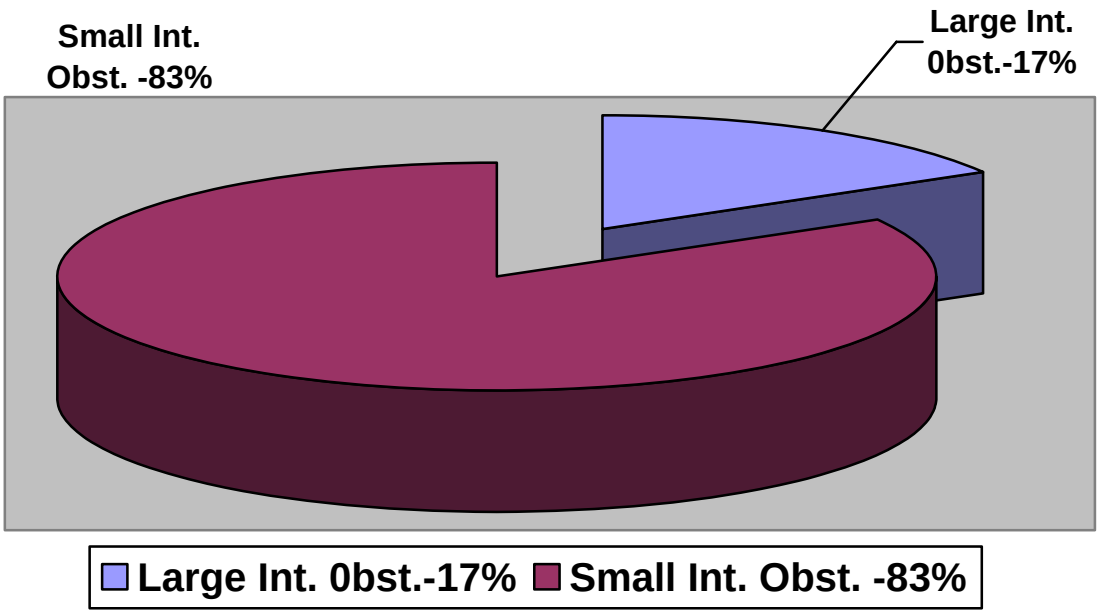
The details of the patient's name, age, sex, IP. No, symptoms at presentation, investigations, intra-operative findings and their outcome were recorded. The observations were tabulated and compared with recent literature and final conclusions derived.

OBSERVATIONS

Total number of patients admitted with Acute Intestinal Obstruction from June 2010 to June 2012 – 100 cases.

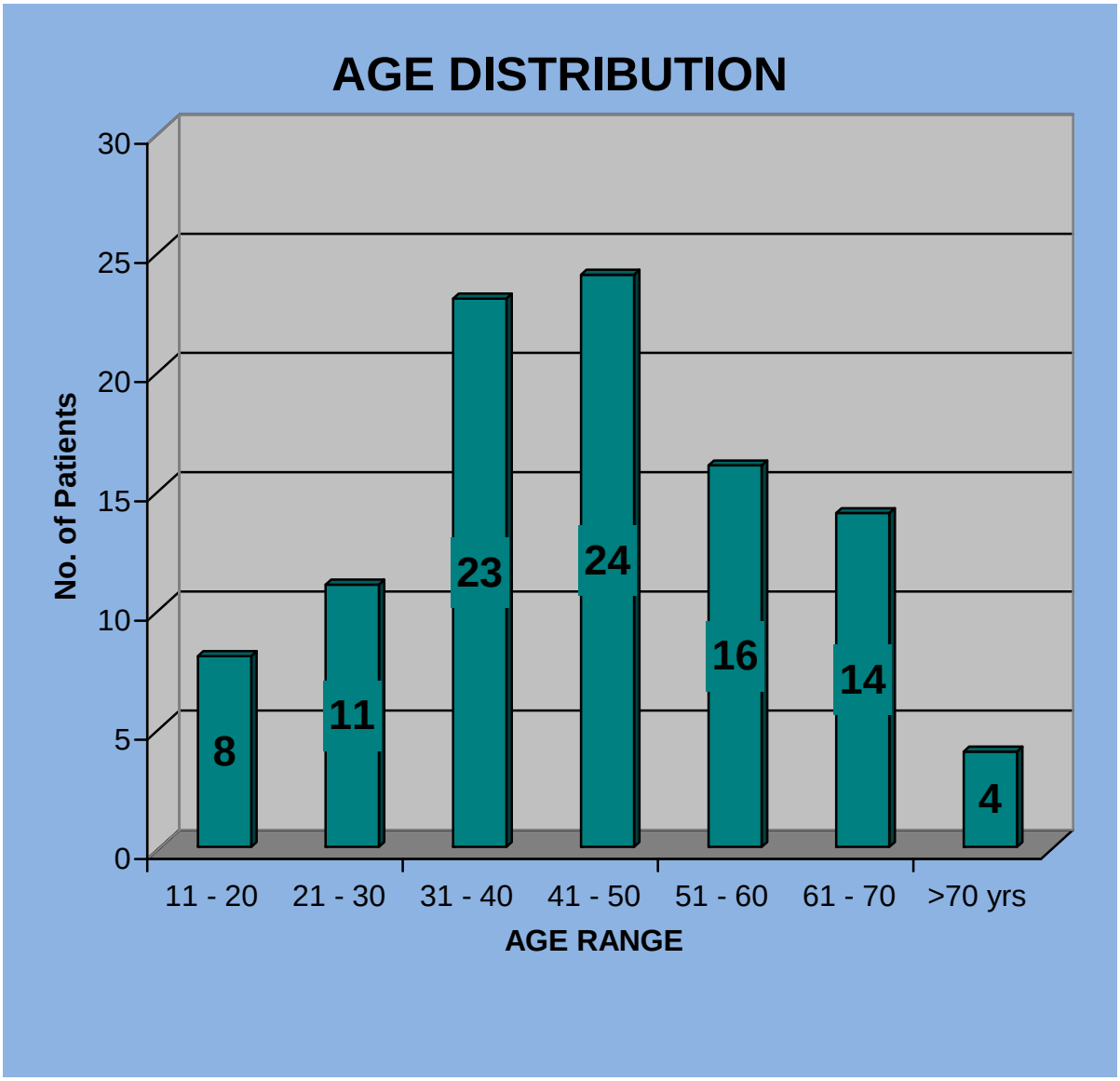
- Large intestine ~ 17
- Small intestine ~ 83

INCIDENCE OF INTESTINAL OBSTRUCTION



AGE DISTRIBUTION

S.No	Age in years	No. of Patients
1.	0 – 10	0
2.	11 – 20	8
3.	21 – 30	11
4.	31 – 40	23
5.	41 – 50	24
6.	51 – 60	16
7.	61 – 70	14
8.	71 – 80	4
9.	81 – 90	0
10.	91 – 100	0
	TOTAL	100



Most common age group affected was between 41 to 50 years.

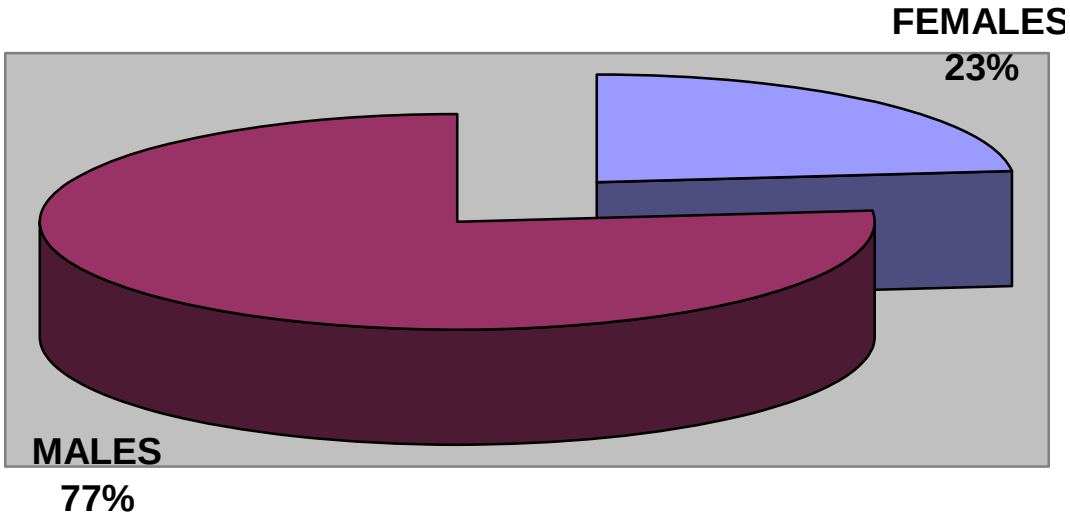
SEX DISTRIBUTION

Males – 77 cases

Females – 23 cases

Males were affected 3.5 times as common as females

SEX DISTRIBUTION

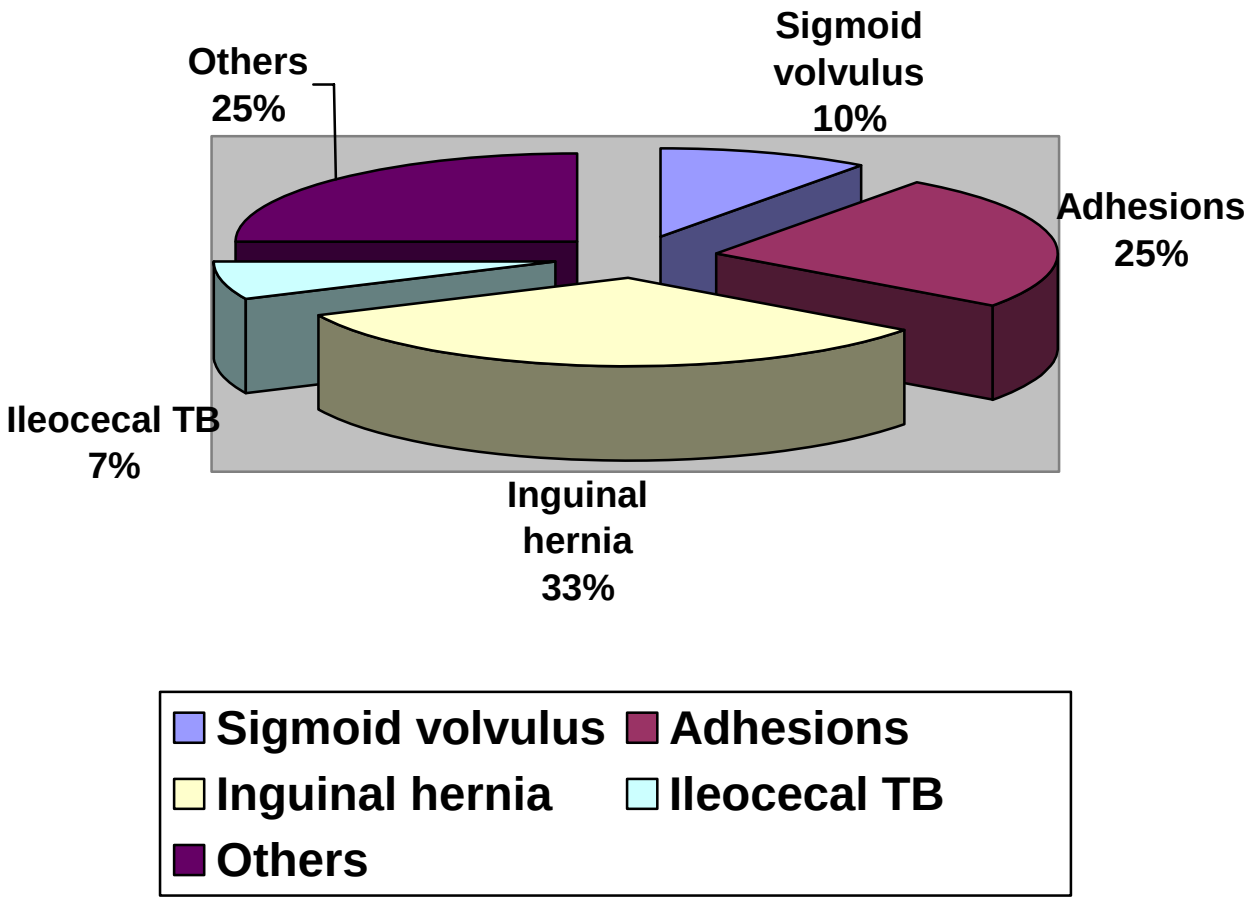


ETIOLOGY

S. No	ETIOLOGY	NO. OF CASES
1.	Obstructed/ Strangulated inguinal hernia	33
2.	Adhesive obstruction	25
3.	Ileocecal Tuberculosis	7
4.	Para umbilical hernia	6
5.	Incisional hernia	3
6.	Femoral hernia	2
7.	Intussusception	1
8.	Meckel's diverticulum	1
9.	Ascending colon / hepatic flexure growth	2
10.	Descending colon growth	1
11.	Sigmoid colon growth	4
12.	Rectum/ anal canal growth	3
13.	Sigmoid volvulus	10
14.	SMA* syndrome	2

*SMA – Superior mesenteric artery syndrome.

ETIOLOGY OF INTESTINAL OBSTRUCTION

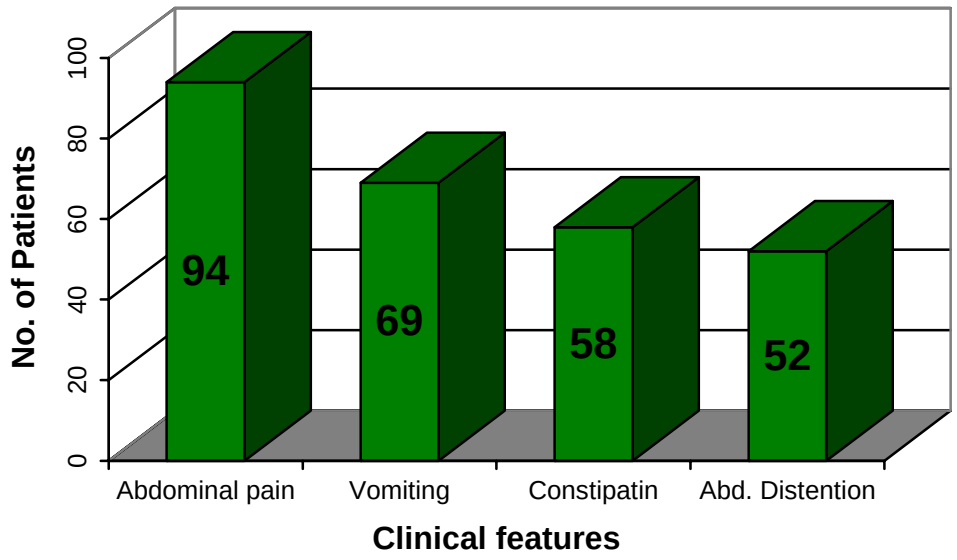


Obstructed inguinal hernia was found to be most common cause followed by adhesive obstruction.

CLINICAL FEATURES

S. No	Clinical features	No. of cases
1.	Abdominal pain	94
2.	Vomiting	69
3.	Constipation	58
4.	Abdominal distention	52

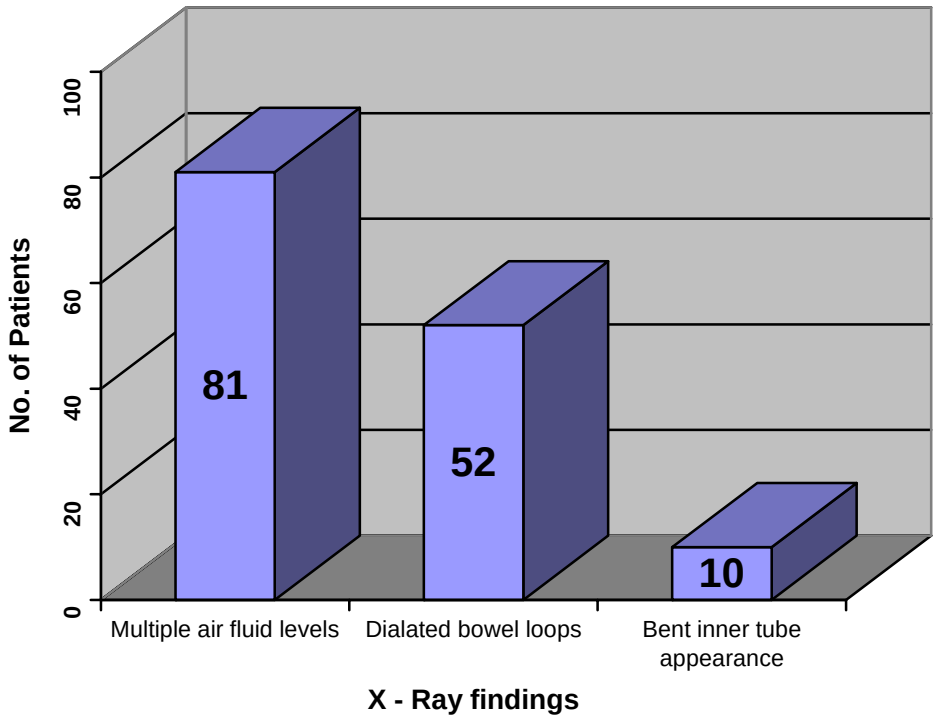
CLINICAL FEATURES



RADIOLAGICAL FINDINGS

S. No	Radiological findings	No. of Patients
1.	Multiple air fluid levels	81
2.	Dilated bowel loops	52
3.	Bent inner tube appearance	10

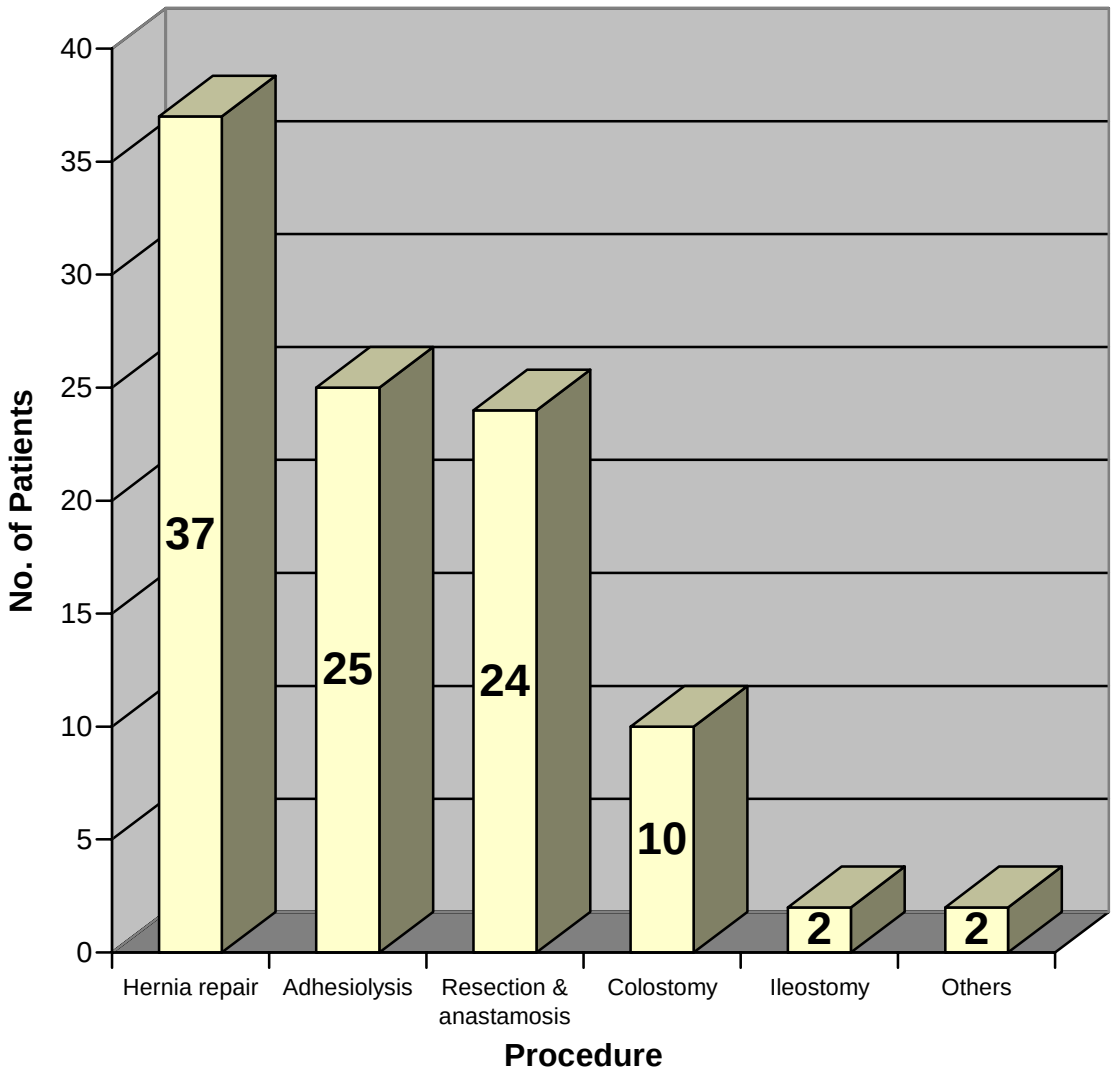
RADIOLOGICAL FINDINGS



SURGICAL TREATMENT

S. No	Procedure	No. of cases
1.	Hernia repair	37
2.	Adhesiolysis	25
3.	Resection and anastomosis	24
4.	Colostomy	10
5.	Ileostomy	2
6.	Others	2

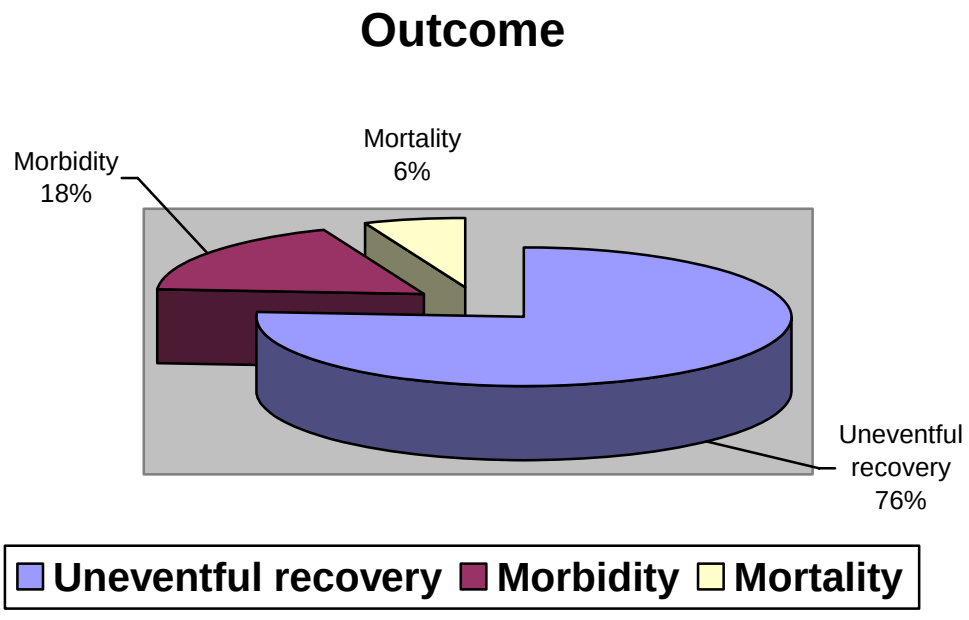
SURGICAL TREATMENT



OUTCOME

In most of the patients post-operative period was uneventful. The outcome was tabled as below,

S. No	Outcome	No. of Patients
1.	Uneventful recovery	76
2.	Morbidity	18
3.	Mortality	6



DISCUSSION

In this study conducted for 2 years from June 2010 – June 2012 in Government Rajaji Hospital, Madurai about 100 patients with acute intestinal obstruction were studied and various data's collected from the study were discussed below.

Site of obstruction:

Of the 100 cases, 83 were due to small bowel obstruction and 17 were due to large bowel obstruction. Hence small bowel was frequently involved than the large bowel. Similar reports are seen in a study conducted by Arshad M. Malik and colleagues in the year 2010. [35]

Age distribution:

Most common age group affected was between 41 – 50 years (24 patients) followed by 31 – 40 years (23 patients). The mean age incidence is 45.5years. The results are comparable to a similar study conducted by Adhikari and colleagues. [35, 36]

Sex distribution:

Of the 100 patients with acute intestinal obstruction 77% were males and 23% were females which is consistent with the sex incidence of similar studies conducted by Miller and colleagues and many others similar studies. [34, 35, 36]

Etiology:

Among the 100 cases, most common cause of acute intestinal obstruction was found to be obstructed/strangulated inguinal hernia which accounted for 33% of the cases. Second most common cause was found to be adhesions which accounted for 25% of cases. Of the 5 cases with strangulation 4 were due to inguinal hernia and 1 was due to incisional hernia. Similar results have been noted in a study conducted by Adhikari Souvik and colleagues in eastern India in 2010. Although post-operative adhesions were found to be the most common cause of obstruction worldwide, in our study inguinal hernias account for most of the cases.[34, 35, 36]

Clinical features:

Most of the cases presented with abdominal pain (94%) followed by vomiting (69%), constipation (58%) and abdominal distention (52%). In a similar study conducted by Markogiamakis and colleagues in 2007 absence of passage of flatus was noted in 90% and feces in 80.6% of patients and abdominal distention was noted in 65.3% of patients. [37]

Radiological findings:

Most common radiological finding was multiple air fluid levels seen in plain X-ray abdomen erect view. This finding was seen in 81 patients followed by dilate bowel loops seen in 52 patients and bent inner tube appearance seen in 10 patients. The observations are comparable to a similar study conducted by Arshad M. Malik and colleagues conducted in the year 2010. [35]

Surgical procedure:

Most common surgical procedure adopted was hernia reduction and repair (37%), which included inguinal, femoral, incisional and paraumblical hernia repairs. Next common procedure was Adhesiolysis (25%) followed by resection and anastomosis (24%) and others. In our study most of patients underwent open surgery although studies conducted by Nagle and colleagues on laparoscopic Adhesiolysis in 2004 states that laparoscopic treatment appears to be effective in selected group of patients. [38]

Outcomes:

Most of the cases recovered without any complications (76%). Infection was the major cause of morbidity and was seen in 18% of patients. Mortality was 6% and was commonly seen in patients with strangulation and increased age. Of 6 deaths 4 were due to sepsis and 2 were due to aspiration. This observation is comparable to a similar study conducted by Adhikari Souvik and colleagues in 2010. [36, 39]

CONCLUSION

This study on acute intestinal obstruction was aimed at studying the age and sex distribution, various etiologies, clinical presentations, different treatment modalities and the outcomes of acute intestinal obstruction.

Acute intestinal obstruction remains to be one of common surgical emergencies. Males are commonly affected mostly during their fourth decades. Obstructed/strangulated inguinal hernia remains to be the most common cause followed by adhesions. They usually present with abdominal pain with multiple air fluid levels in their X-ray abdomen erect view. The initial management of patients with acute intestinal obstruction should focus on aggressive fluid resuscitation, decompression of the obstructed bowel, and on prevention of aspiration. Surgery remains to be the cornerstone of treatment.

Earlier diagnosis and timely intervention are associated with excellent prognosis. Delayed diagnosis leading to strangulation and increased age are associated with poor outcomes.

PROFORMA

Name : **IP No:**

Age & Sex: **Unit :**

Occupation:

Present History:

H/O Abdominal pain

H/O Vomiting

H/O Constipation

H/O Abdominal distention

Past History:

Personal History:

Family History:

General Examination:

- ✓Signs of dehydration
- ✓Blood pressure
- ✓Pulse rate

Examination of Abdomen:

Inspection -

Palpation -

Percussion -

Auscultation –

PR & PV –

Examination of Other systems:

Cardiovascular system –

Respiratory system –

Central nervous system –

Investigations:

Hb%	TC
DC	ESR
RBS	Urea
Creatinine	CXR PA View
X-ray abdomen erect view	ECG

Management:

Follow up:

BIBLIOGRAPHY

1. Sabiston Textbook of Surgery, 18th edition, 2008; Volume 2, Small intestine, Page no 1278-1297; Colon and Rectum, Page no 1348-1373.
2. Last's Anatomy regional and applied, 10th Edition, 2001, Chapter 5,

   Part 6, Page no: 246 – 252.
3. Gray's Anatomy, 39th edition, 2005. Gastrointestinal tract- Small intestine and Large intestine, Page no 1157-1205.
4. Skandalakis Surgical Anatomy and Technique, 2nd edition, 2000; Small Intestine page no 425- 432 and Colon and Anorectum, Page no 457-480.
5. B D Chaurasia's Human Anatomy, 4th edition, 2006; Volume 2, The Intestines, Page no 245-271.
6. Lee Mc Gregor's synopsis of surgical anatomy, 1995, 12th Edition,

   Chapter 3, Page no: 22 -23.

7. Maingot's Abdominal Operation's, 11th edition, 2007; Volume 1, Bowel obstruction, Page no 479- 505.
8. SRB's Manual of Surgery, 3rd edition, 2010; Intestinal obstruction, Page no 851- 872.
9. Bailey and Love's Short Practice of Surgery, 25th edition, 2008; Chapter 66, Intestinal Obstruction, Page no 1188-1203.
10. Schwartz's Principles of Surgery, 9th edition, 2010; Small intestine, Page no 979-991; Colon, Rectum, and Anus, Page no 1015-1019 and 1041-1057.
11. Hamilton Bailey's Emergency Surgery, 13th edition, 2009; Intestinal obstruction: general principles, Small bowel obstruction, large bowel obstruction, Page no 420- 435.
12. Lu RH, Chang TM, Yen MH et al. Involvement of superoxide anion in the pathogenesis of simple mechanical intestinal obstruction. J Surg Res 2003; 115:184–190.
13. Miedema BW, Johnson JO. Methods for decreasing postoperative gut dysmotility. Lancet Oncol 2003; 4:365–372.

14. Hellebrekers BW, Trimbos-Kemper GC, Bakkum EA et al. Short-term effect of surgical trauma on rat peritoneal fibrinolytic activity and its role in adhesion formation. Thromb Haemost 2000; 84:876–881.
15. Wilson MS, Ellis H, Menzies D et al. A review of the management of small bowel obstruction. Members of the Surgical and Clinical Adhesions Research Study (SCAR). Ann R Coll Surg Engl 1999; 81:320–328.
16. Sulaiman H, Gabella G, Davis C et al. Presence and distribution of sensory nerve fibers in human peritoneal adhesions. Ann Surg 2001; 234:256–261.
17. Di Zerega GS, Campeau JD. Peritoneal repair and post-surgical adhesion formation. Hum Reprod Update 2001; 7:547–555.
18. Mucha P Jr. Small intestinal obstruction. Surg Clin North Am 1987; 67:597–620.
19. Assalia A, Schein M, Kopelman D et al. Therapeutic effect of oral Gastrografin in adhesive, partial small-bowel

obstruction: a prospective randomized trial. Surgery 1994; 115:433–437.

20. Choi HK, Chu KW, Law WL. Therapeutic value of Gastrografin in adhesive small bowel obstruction after unsuccessful conservative treatment: a prospective randomized trial. Ann Surg 2002; 236:1-6.
21. Feigin E, Seror D, Szold A et al. Water-soluble contrast material has no therapeutic effect on postoperative small-bowel obstruction: results of a prospective, randomized clinical trial. Am J Surg 1996; 171:227–229.
22. Okada T, Yoshida H, Iwai J et al. Pulsed Doppler sonography for the diagnosis of strangulation in small bowel obstruction. J Pediatr Surg 2001; 36:430–435.
23. Cozza S, Ferrari FS, Stefani P et al. Ileal occlusion with strangulation: importance of ultrasonography findings of the dilated loop with intraluminal fluid-fluid resulting from sedimentation. Radiol Med (Torino) 1996; 92:394–397.

24. Gowen GF. Long tube decompression is successful in 90% of patients with adhesive small bowel obstruction. Am J Surg 2003; 185:512 -515.

25. Brolin RE. The role of gastrointestinal tube decompression in the treatment of mechanical intestinal obstruction. Am Surg 1983;49:131–137.

26. Fleshner PR, Siegman MG, Slater GI et al. A prospective, randomized trial of short versus long tubes in adhesive small-bowel obstruction. Am J Surg 1995; 170:366–370.

27. Fevang BT, Jensen D, Svanes K et al. Early operation or conservative management of patients with small bowel obstruction? Eur J Surg 2002; 168:475–481.

28. Baerga-Varela Y. Small bowel obstruction. In: Kelly KA, Sarr MG, Hinder RA (Eds). Mayo Clinic Gastrointestinal Surgery, 1st ed. Philadelphia, PA: Saunders; 2004:421–437.

29. Chen SC, Chang KJ, Lee PH et al. Oral Urografin in postoperative small bowel obstruction. World J Surg 1999; 23:1051-1054.

30. Farquharson's Textbook of Operative General Surgery, 9th edition; Surgery of Intestinal obstruction, Page no 409- 417.

31. Hughes SJ. Large bowel obstruction. In: Bland KI (ed). The Practice of General Surgery, 1st ed. Philadelphia, PA: Saunders; 2002:473–477.

32. Suzuki K, Umehara Y, Kimura T. Elective laparoscopy for small bowel obstruction. Surg Laparosc Endosc Percutan Tech 2003; 13:254–256.

33. Baron TH, Kozarek RA. Endoscopic stenting of colonic tumours. Best Pract Res Clin Gastroenterol 2004; 18:209–229.

34. Miller G, Boman J, Shrier I, Gordon PH. Etiology of small bowel obstruction. Am J Surg 2000; 180(1):33-6.

35. Arshad M. Malik, Madiba Shah, Rafique Pathan, Krishan Sufi. Pattern of Acute Intestinal Obstruction: Is there a change in underlying etiology? Saudi J Gastroenterol 2010; 16(4):272-274.

36. Adhikari Souvik, Mohammed Zahid Hossain, Das Amitabha, Mitra Nilanjan, Ray Udipta. Etiology and outcome of Acute

Intestinal Obstruction: A Review of 367 patients in Eastern India. Saudi J Gastroenterol 2010:16(4) 285-287.

37. Markogiamakis H, Messaris E, Darda Manis D, Pararas n, Tzertzemelis, Gianopoulos P, Larentzakis A, Lagoudianakis E, Manouras, Bramis I. Acute mechanical bowel obstruction : Clinical presentation, etiology, management and outcome. World J Gastroenterol 2007:2113(3); 432-7.

38. Nagle A, Ujiki M, Denham W, et al: Laparoscopic Adhesiolysis of adhesions for small bowel obstruction. Am J Surg 187:464-470, 2004.

39. Chen XZ, Wei T, Jiang K, Yang K, Zhang B, Chen ZX, Chen JP, Hu JK. Etiological factors and mortality of Acute Intestinal Obstruction: A Review of 705 cases. Zhong Xi Yi Jie He Xue Bao 2008;6(10):1010-6.

MASTER CHART

S. No	Name	IP. No	AGE	SEX	CLINICAL PRESENTATION				X – RAY ABDOMEN ERECT	DIAGNOSIS	PROCEDURE
					ABD. PAIN	VOMITING	CONSTIPATION	DISTENTION			
1.	Govindraj	51332	50	M	+	-	+	+	BITA	Sigmoid Volvulus	Hartmann's procedure
2.	Pappathi	65131	50	F	+	+	+	+	MAFL, DBL	Adhesive Obstruction	Adhesiolysis
3.	Muniyandi	85521	40	M	+	+	+	+	MAFL	TB Abdomen with Ileal stricture	Resection and anastomosis
4.	Sundararajan	85320	65	M	+	+	+	+	MAFL	Obstructed Inguinal Hernia	Hernioplasty
5.	Muniyan	18617	50	M	+	-	+	-	MAFL, DBL	SMA Occlusion	Resection and anastomosis

6.	Subash	19215	16	M	+	+	+	+	MAFL	Adhesive obstruction	Adhesiolysis
7.	Sesuraj	34865	42	M	+	+	+	+	MAFL	Ileocecal TB	Resection and Ileo-transverse anastomosis
8.	Natchipillai	35101	58	M	+	+	+	-	MAFL	Ileocecal TB	Limited resection and anastomosis
9.	Maharajan	36834	50	M	+	-	+	+	MAFL	Obstructed Inguinal Hernia	Hernioplasty
10.	Thangavel	42441	65	M	+	+	+	-	DBL	Adhesive obstruction	Adhesiolysis
11.	SundarRajan	10050	36	M	-	-	+	+	BITA	Sigmoid Volvulus	Resection and anastomosis
12.	Kundumalai	29505	58	M	+	+	-	-	MAFL	Adhesive Obstruction	Adhesiolysis
13.	Bhakyamani	34424	40	F	+	+	-	-	MAFL	Intussusception	Ileal resection and anastomosis
14.	Arumugam	36542	70	M	-	+	-	-	DBL	Strangulated Inguinal Hernia	Resection and anastomosis

15.	Duraisamy	52180	42	M	+	-	+	+	BITA	Sigmoid Volvulus	Sigmoid Resection and anastomosis
16.	Soundaravalli	67811	35	F	+	-	+	+	MAFL, DBL	Ca Sigmoid colon	Hartmann's procedure
17.	Jothi	77379	42	F	+	+	-	-	MAFL	Adhesive obstruction	Adhesiolysis
18.	Uma Sangareshvari	80933	19	F	+	+	-	-	MAFL	TB abdomen	Adhesiolysis
19.	Maheshwari	84012	35	F	+	-	+	+	BITA	Sigmoid Volvulus	Hartmann's procedure
20.	Murugan	82012	13	M	+	+	+	+	BITA	Sigmoid Volvulus	Sigmoid resection and anastomosis
21.	Murugesan	19453	33	M	-	-	+	+	MAFL	Sigmoid growth	Hartmann's procedure
22.	Saravana kumar	14210	22	M	+	+	-	-	DBL	Adhesive Obstruction	Adhesiolysis
23.	Muthupillai	82577	48	F	+	+	+	-	MAFL, DBL	Obstructed Inguinal Hernia	Herniorrhaphy

24.	Balakrishnan	93354	32	M	+	+	-	+	MAFL	Obstructed Inguinal Hernia	Herniorrhaphy
25.	Kesavan	01207	40	M	+	+	-	-	MAFL, DBL	Obstructed Inguinal Hernia	Herniorrhaphy
26.	Kadamban	01302	58	M	+	+	-	-	MAFL	Adhesive Obstruction	Adhesiolysis
27.	Palanisamy	09108	30	M	+	+	-	+	DBL	Strangulated Inguinal Hernia	Resection and anastomosis
28.	Vellaisamy	12260	35	M	+	+	-	-	MAFL	Adhesive Obstruction	Adhesiolysis
29.	Deivani	12298	58	F	+	+	-	-	MAFL, DBL	Adhesive Obstruction	Adhesiolysis
30.	Ponraj	35426	63	M	-	-	+	+	MAFL, DBL	Sigmoid growth	Diversion colostomy
31.	Kumaravel	35373	28	M	+	+	-	-	MAFL	Adhesive Obstruction	Adhesiolysis
32.	Ramamoorthy	36514	40	M	+	+	-	-	MAFL, DBL	Adhesive Obstruction	Adhesiolysis
33.	Gopi	43606	23	M	+	+	-	-	MAFL	Adhesive	Adhesiolysis

										Obstruction	
34.	Jeyapal	56316	62	M	+	+	-	-	MAFL, DBL	Obstructed Inguinal Hernia	Herniorrhaphy
35.	Saraswathy	69238	60	F	+	+	-	-	MAFL	Adhesive Obstruction	Adhesiolysis
36.	Pathrakali	82755	40	F	+	-	+	+	BITA	Sigmoid Volvulus	Sigmoid resection and anastomosis
37.	Kalaivani	05872	29	F	+	+	-	-	MAFL, DBL	Adhesive Obstruction	Adhesiolysis
38.	Amirtham	06918	65	F	+	+	-	-	MAFL	Adhesive Obstruction	Adhesiolysis
39.	Selvaraj	10644	52	M	+	+	-	+	MAFL	Adhesive Obstruction	Adhesiolysis
40.	Venkatesh	14017	40	M	+	+	-	+	MAFL	Adhesive Obstruction	Adhesiolysis
41.	Chinnaadaikan	17522	55	M	+	-	+	-	DBL	SMA occlusion	Resection and anastomosis
42.	Arjunan	22472	55	M	+	+	+	+	MAFL, DBL	Obstructed Inguinal Hernia	Herniorrhaphy

43.	Velusamy	27731	42	M	+	+	+	-	MAFL, DBL	Obstructed Paraumblical Hernia	Hernia repair
44.	Arunprakash	46958	20	M	+	+	+	-	DBL	Adhesive Obstruction	Adhesiolysis
45.	Vetriarasan	42846	69	M	+	-	+	-	MAFL	Descending colon growth	Loop Colostomy
46.	Mohan	44737	28	M	+	+	-	+	MAFL, DBL	Adhesive Obstruction	Adhesiolysis
47.	Pavalakodi	45637	35	F	+	+	+	+	MAFL, DBL	Obstructed Incisional Hernia	Incisional Hernia Repair
48.	Arumugam	37885	25	M	+	+	-	+	MAFL	Meckel's Diverticular Obstruction	Segmental resection and anastomosis
49.	Mani	41010	50	M	+	+	+	-	MAFL, DBL	Obstructed Inguinal Hernia	Hernioplasty
50.	Janakiraman	44507	32	M	+	+	+	-	MAFL, DBL	Obstructed Paraumblical Hernia	Hernia repair
51.	Karthick	44532	67	M	+	+	+	+	MAFL	Obstructed Inguinal Hernia	Hernioplasty

52.	Muthukrishnan	49318	66	M	+	-	+	+	BITA	Sigmoid Volvulus	Sigmoid resection and anastomosis
53.	Paramesan	52379	50	M	+	+	+	+	MAFL, DBL	Obstructed Paraumblical Hernia	Anatomical hernia repair
54.	Kalidas	52490	20	M	+	-	+	-	MAFL, DBL	Obstructed Inguinal Hernia	Herniorrhaphy
55.	Arumugasamy	55593	45	M	+	+	-	-	MAFL, DBL	Adhesive Obstruction	Adhesiolysis
56.	Velusamy	56522	39	M	+	+	+	+	MAFL, DBL	Obstructed Inguinal Hernia	Hernioplasty
57.	Kannan	56987	49	M	+	+	-	+	MAFL	Obstructed Inguinal Hernia	Herniorrhaphy
58.	Ashwin	58765	14	M	+	+	-	-	MAFL	Post appendectomy adhesions	Adhesiolysis
59.	Seemaan	58986	40	M	+	+	-	-	MAFL, DBL	Adhesive Obstruction	Adhesiolysis
60.	Alagammal	63867	80	F	+	-	+	+	BITA	Sigmoid Volvulus	Sigmoid resection and anastomosis
61.	Irulappan	78621	45	M	+	+	+	+	MAFL,	Obstructed	Herniorrhaphy

									DBL	Inguinal Hernia	
62.	Mani	94410	45	M	+	-	+	+	MAFL, DBL	Obstructed Inguinal Hernia	Herniorrhaphy
63.	Narayanan	80542	48	M	+	+	+	-	MAFL, DBL	Obstructed Inguinal Hernia	Herniorrhaphy
64.	Ramamoorthy	01120	59	M	+	+	-	+	MAFL, DBL	Obstructed Inguinal Hernia	Herniorrhaphy
65.	Raman	05718	30	M	+	+	-	+	MAFL	Obstructed Inguinal Hernia	Herniorrhaphy
66.	Seeniammal	08278	79	F	-	-	+	+	MAFL, DBL	Rectal growth	Hartmann's procedure
67.	Ganesan	06148	40	M	+	-	+	+	BITA	Sigmoid Volvulus	Hartmann's procedure
68.	Saravanan	27214	42	M	+	-	+	+	MAFL, DBL	Obstructed Inguinal Hernia	Hernioplasty
69.	Krishnammal	72339	45	F	-	-	+	+	MAFL, DBL	Rectal growth	Loop colostomy
70.	Santhan	75745	20	M	+	-	+	-	MAFL, DBL	Strangulated Inguinal Hernia	Segmental resection and

											anastomosis
71.	Angammal	75718	30	F	+	+	-	+	MAFL, DBL	Obstructed paraumblical hernia	Hernia reduction and repair
72.	Periyasamy	87355	78	M	+	+	+	-	MAFL	Obstructed Inguinal Hernia	Hernioplasty
73.	Ayyanar	82433	45	M	+	+	-	+	MAFL, DBL	Obstructed Inguinal Hernia	Herniorrhaphy
74.	Thirumeni	06403	48	F	+	+	-	-	MAFL	Obstructed Incisional Hernia	Hernia reduction and repair
75.	Muthumani	06647	29	M	+	-	+	+	MAFL, DBL	Ascending colon growth	Right hemi-colectomy
76.	Narasimman	28933	70	M	+	+	-	-	MAFL	Obstructed Inguinal Hernia	Herniorrhaphy
77.	Ganesan	30748	50	M	+	-	-	+	MAFL, DBL	Hepatic flexure growth	Ileo-transverse anastomosis
78.	Karupayamma	32705	55	F	+	-	+	+	MAFL, DBL	Rectal growth	Sigmoid loop colostomy
79.	Nagappan	38528	65	M	+	-	+	-	MAFL, DBL	Obstructed Inguinal Hernia	Hernioplasty

80.	Gurusamy	36619	15	M	+	+	+	+	MAFL	Obstructed Inguinal Hernia	Hernioplasty
81.	Baskar	45651	52	M	+	-	+	+	MAFL, DBL	Obstructed Inguinal Hernia	Hernioplasty
82.	Alavutheen	55729	67	M	+	+	+	+	MAFL	Obstructed Inguinal Hernia	Hernioplasty
83.	Ammavasai	57573	55	M	+	+	-	-	MAFL, DBL	Tuberculous Ileal stricture	Segmental resection and anastomosis
84.	Sundharam	60818	31	M	+	-	+	+	MAFL	Obstructed Inguinal Hernia	Hernioplasty
85.	Maiyayee	62759	45	F	+	+	+	-	MAFL, DBL	Strangulated Femoral hernia	Segmental resection and anastomosis
86.	Muthulakshmi	62912	48	F	+	-	+	+	BITA	Sigmoid Volvulus	Sigmoid resection and anastomosis
87.	Pitchaimuthu	66450	50	M	+	+	+	-	DBL	Strangulated paraumblical hernia	Segmental resection and anastomosis
88.	Nallathambi	79637	70	M	+	+	-	-	MAFL, DBL	Obstructed paraumblical hernia	Hernia reduction and repair
89.	Kadarkaraiam	86147	55	F	+	+	+	-	MAFL,	Obstructed	Femoral Hernia

	mal								DBL	Femoral Hernia	repair
90.	Jeyaraman	88906	55	M	+	+	-	+	DBL MAFL	Obstructed Inguinal Hernia	Hernioplasty
91.	Jeyaram	91971	40	M	+	-	+	+	MAFL	Obstructed Inguinal Hernia	Hernioplasty
92.	Subramani	63641	40	M	+	+	+	-	DBL	Strangulated Incisional hernia	Segmental resection and anastomosis
93.	Chandran	23272	34	M	+	+	-	-	MAFL	Adhesive obstruction	Adhesiolysis
94.	Kathiresan	31559	29	M	+	+	+	+	MAFL	Obstructed Inguinal Hernia	Hernioplasty
95.	Sinthamani	36296	64	M	+	-	+	+	MAFL, DBL	Obstructed Inguinal Hernia	Hernioplasty
96.	Pitchaiammal	38139	55	F	+	+	-	-	DBL	Adhesive obstruction	Adhesiolysis
97.	Anitha	39823	35	F	+	+	-	-	MAFL	Ileocecal TB	Adhesiolysis
98.	Ravi	36858	66	M	+	-	+	+	MAFL, DBL	Sigmoid colon growth	Sigmoid resection and anastomosis

99.	Parameshwari	36950	40	F	+	+	-	-	MAFL	Ileocecal TB	Limited resection and anastomosis
100.	Ramesh	37086	60	M	+	+	-	-	MAFL	Adhesive obstruction	Adhesiolysis

MAFL – Multiple Air Fluid Levels, DBL – Dialated Bowel Loops, BITA- Bent Inner Tube Appearance,

M – Males, F- Females.

A Study of

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A Study of ACUTE INTESTINAL OBSTRUCTION Dissertation submitted to THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY CHENNAI – 600 032 With fulfillment of the Regulations For the Award of the Degree of M.S. GENERAL SURGERY (BRANCH – I) DEPARTMENT OF GENERAL SURGERY GOVERNMENT RAJAJI MEDICAL COLLEGE AND HOSPITAL MADURAI – 625 020 MARCH – 2013 CERTIFICATE This is to certify that this dissertation in "ACUTE INTESTINAL OBSTRUCTION" is a work done` by DR. TAMIL MUTHU.M, under my guidance during the period 2010-2012. This has been submitted in partial fulfillment of the requirements for the award of M.S. Degree in General Surgery (Branch – I) by the Tamil Nadu Dr. M.G.R. Medical University, Chennai – 32...